# PREVALENCE OF MESENTERIC VASCULAR DISEASE IN PATIENTS WITH CORONARY ARTERY DISEASE

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# INTRODUCTION

## Mesenteric Ischemia

Mesenteric artery ischemia ensues following reduced perfusion of the intestines. It is usually secondary to narrowing or blockage of one or more of the three mesenteric arteries, the major arteries that supply the gastrointestinal tract. Uncommonly ischemia can be result of reduced perfusion in the absence of occlusion of mesenteric arteries. While acute mesenteric ischemia has a dramatic presentation, chronic mesenteric ischemia has an insidious course. The

# CERTIFICATE

This is to certify that this dissertation titled

**"PREVALENCE OF MESENTERIC VASCULAR DISEASE IN PATIENTS WITH CORONARY ARTERY DISEASE"**

is the bonafide work done by **Dr. Gautham K.,** Post Graduate student (2011 – 2014) in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under my direct guidance and supervision, in partial fulfillment of the regulations of The Tamil Nadu Dr. M.G.R Medical University, Chennai for the award of M.S., Degree (General Surgery) Branch - I, Examination to be held in April 2014.

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# DECLARATION

I, **DR. K. GAUTHAM** solemnly declare that this dissertation titled "**PREVALENCE OF MESENTERIC VASCULAR DISEASE IN PATIENTS WITH CORONARY ARTERY DISEASE**" is a bonafide work done by me in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under the guidance and supervision of my unit chief.

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# ABSTRACT


## Prevalence of Mesenteric Vascular Disease in Coronary Artery Disease Patients

### Introduction

Atherosclerosis is a global phenomenon affecting the primarily the medium sized arteries irrespective of the vascular bed. Hence the involvement of a vascular site at one site predisposes involvement of another distant site. The prevalence chronic mesenteric ischemia is often underestimated because of its non-specific symptomatology. Patient related and physician related are responsible for the delay in the diagnosis till the stage of bowel infarction when there is high morbidity and mortality. Paucity of studies on the natural course of disease further aggravates the situation. Given the prevalence of coronary artery disease is very high, the same population should also be predisposed for chronic mesenteric ischemia.

### Materials and Methodology

110 patients, who underwent coronary angiogram for evaluation of suspected coronary artery disease, were subjected to mesenteric angiogram at the same sitting.

### Results

Out of 110 patients, 7 had normal coronary study. The remaining 103 patients were evaluated for synchronous mesenteric artery involvement. 44 out of 103 (42.7%)

had mesenteric involvement out of which 19 had indications for revascularization ($p<0.05$). There was no difference between the genders. Higher age group (>65 years), diabetic and cases of known peripheral vascular disease were predisposed for mesenteric artery involvement ($p<0.05$). Superior mesenteric artery was the most common artery involved (72.2%). There was no correlation between the severity of the coronary disease and mesenteric disease. However involvement of left anterior descending artery had positive association with mesenteric artery involvement.

**Conclusion**

The prevalence of mesenteric artery disease is common among the coronary artery disease patients. The probability increases in elderly, diabetic and patients with prior history of peripheral vascular disease. Further studies with longer follow-up can help in understanding the natural course of the disease and thereby provide better idea regarding the time of intervention.

**Keywords**

Mesenteric vascular disease, coronary artery disease, atherosclerosis, angiogram, chronic mesenteric ischemia

# INTRODUCTION

## Mesenteric Ischemia

Mesenteric artery ischemia ensues following reduced perfusion of the intestines. It is usually secondary to narrowing or blockage of one or more of the three mesenteric arteries, the major arteries that supply the gastrointestinal tract. Uncommonly ischemia can be result of reduced perfusion in the absence of occlusion of mesenteric arteries. While acute mesenteric ischemia has a dramatic presentation, chronic mesenteric ischemia has an insidious course. The fact that the natural course of chronic mesenteric ischemia is gradual and non-specific at early stages makes diagnosis of chronic mesenteric ischemia delayed by months or even years. Patient and physician related factors play a vital role in the failure to recognize the initial vague symptoms of mesenteric insufficiency invariably till the point of no return of bowel infarction when acute ischemia sets in (1).

## Anatomy

The abdominal aorta measures 10 cms extending from T12 to lower one-third of the body of L4 (2). Given it is the principal artery supplying the multiple intra-abdominal organs, the large size of the branches

supplying the major abdominal viscera results in marked drop in aortic diameter through its descend. Within the 10 cm course the aorta which is 25 mm at the level of 11th rib becomes 19 mm in diameter at its bifurcation (3).

The fact that, during embryogenesis, primitive gut lies anterior to the aorta makes the arteries that supply the gut ventrally placed. In contrast the arteries supplying the kidney and the gonads, retroperitoneal in origin, are laterally placed.

The three principal arteries supplying the intra-abdominal gastrointestinal tract are celiac trunk, superior and inferior mesenteric artery (4). Anatomical variation of these vessels is common with celiac trunk being most prone. Recognition of this fact is important during major abdominal and laparoscopic surgeries to avoid catastrophes (5).

**Embryology**

Ventral segmental arteries emerge from the primitive ventral aorta. Most of these ventral segmental arteries regress with persistence of the $10^{th}$, $13^{th}$ and $21^{st}$ branches forming the celiac axis, superior mesenteric artery and inferior mesenteric artery respectively. Deviations in the visceral

arterial anatomy results from disparity in the regression of the primitive ventral aorta and its segmental branches.

**Celiac Axis**

Celiac axis arises from the abdominal aorta at the level of L1. It is bordered superiorly by median arcuate ligament and inferiorly by the pancreas. The three branches from common trunk include the left gastric, splenic, and common hepatic arteries. However, multiple variations of the true “trifurcation” can exist.

**Superior Mesenteric Artery**

Superior mesenteric artery arises 1 centimeter distal to the celiac trunk from the aorta. It usually arises at an angle of 36–56 degrees with the aorta (6). At its origin the SMA lies superior to the uncinate process of the pancreas and the third portion of the duodenum. The inferior pancreatico-duodenal artery and middle colic artery are first two significant branches and play crucial role in collateral supply. The small bowel is supplied by right colic, ileocolic, jejunal and ileal branches as the SMA courses within the mesentery.

## Inferior Mesenteric Artery

Inferior mesenteric artery, the artery of the hindgut, arises at the level of third lumbar vertebra. It divides into sigmoidal branches and the left colic artery supplying the hindgut derivatives.

## Collateral Circulation

The rich collateral circulation among the three visceral arteries make them relatively immune to ischemia. However absence of these make the gut highly vulnerable to infarction. Various named collateral branches have been described in the literature (7).

*Between Celiac axis and Superior Mesenteric artery*

- Rio Branco arcade – Right pancreatico-duodenal
- Býhler arcade – Left pancreatico-duodenal

*Between Superior and Inferior Mesenteric artery*

- Arc of Riolan (meandering mesenteric artery) - middle and left colic arteries (8)
- Marginal artery of Drummond - distal superior and inferior mesenteric arteries
- Villemin arcade - proximal superior and inferior mesenteric arteries (9)

There are various other unnamed collaterals notable of which is multiple connections between superior rectal artery, which originates from the inferior mesenteric artery, and the middle and inferior rectal arteries that are branches of the internal iliac system.

**Pathophysiology of mesenteric ischemia**

The blood supply to the gastrointestinal tract is dynamic. In contrary to the blood supply of the important viscera like the brain, heart and renal which is vital and cannot be compromised at any condition, the blood supply to the intestines typifies the demand-supply physiology. After food ingestion, demand causes increased intestinal blood flow mediated by various chemicals. The increase in blood flow peaks at 30 to 90 minutes after ingestion (10). The nature of the rise and sustenance of the increased blood supply depends on the size and the composition of the meal (11). On weight basis, fat produces the greatest hyperemia. The contribution of protein and carbohydrate to postprandial intestinal hyperemia though minimal can become significant in the presence of mesenteric insufficiency (12). The small bowel and pancreas are the major beneficiaries with minor increase to the stomach and colon (13). This is demonstrable by the marked increase in end-diastolic flow velocities in the superior mesenteric artery compared to the celiac axis.

The postprandial hyperemic response is attenuated in the presence of a hemodynamically significant arterial stenosis. This relative imbalance leads the accumulation of metabolites leads to postprandial pain or “mesenteric angina” (14).

**Causes of mesenteric ischemia**

Stenosis or occlusion of the mesenteric arterial circulation forms the predominant causative factor in the development of chronic mesenteric ischemia (15). Atherosclerosis is the leading cause chronic mesenteric ischemia. Other causes include fibromuscular disease, aortic dissection, isolated SMA dissection and neurofibromatosis. Collagen vascular diseases and autoimmune disorders such as rheumatoid arthritis, Takayasu's arteritis, giant cell arteritis, polyarteritis nodosa, Buerger's disease and systemic lupus have also been implicated in chronic gastrointestinal ischemia. Given the widespread involvement of the arterial system, primary or secondary thrombosis of the superior mesenteric vein secondary to generalized hypercoagulable states or local pathologies such as abdominal malignancies; and inflammatory conditions, like pancreatitis account for <10% of all CMI cases (15). Apart from atherosclerosis the other etiologies collectively are less significant. However in the presence of appropriate clinical setting,

investigation needs to be performed to rule out these uncommon etiologies.

**Chronic Mesenteric Ischemia**

The true incidence of chronic mesenteric ischemia is difficult to ascertain because of its non-specific presentation at initial stages. The reported incidence is derived from various case series of treated patients which may not reflect at real picture (1). The underlying pathophysiology of chronic mesenteric ischemia is presence of adequate vascular supply at baseline and failure to achieve postprandial hyperemic intestinal blood flow. There are various factors that prevent the understanding the natural course of the disease. The rich collateral circulation of gastrointestinal tract provides adequate cushion for the vascular insufficiency to remain asymptomatic until advanced stage. Physician related factor plays an important role since the disease is not suspected in isolation leading to delay in the diagnosis. Once the disease is diagnosed, by demonstration of arterial occlusion and symptomatology, patients undergo revascularization precluding characterization of the course of the disease. All these factors make decision making tough as to enumerate guidelines regarding the management of chronic mesenteric insufficiency especially in

asymptomatic individuals. Eventually death results from inanition or bowel infarction.

Abdominal pain is usually the presenting symptom often periumbilical with radiation to back. It is usually dull in character. It occurs 15 to 30 minutes after eating and lasts 1 to 3 hours consistent with the underlying pathophysiology. The cause of the pain has been attributed to various ischemic mediators (16). The pain progresses from intermittent pain related with certain types or quantities of food to persistent, unremitting pain that likely forebodes bowel infarction. Physiological adaptation and patient adaptive strategies like diet modification causes partial relief.

These strategies results in profound weight loss. The term ‘sitophobia’ denotes the condition where the patient fears food intake in order to avoid post prandial pain. The contribution of malabsorption to weight is found to be insignificant with inadequate nutritional intake being the primary cause (17). Patients can either have constipation due to food avoidance or may present with diarrhea.

Physical examination is not particularly helpful in terms of identifying chronic mesenteric ischemia. Patients may have evidence of systemic vascular disease and abdominal bruits. The mere absence of systemic

vascular disease does not exclude the diagnosis since the vascular disease may be confined to the central aortic region (18).

**Acute Mesenteric Ischemia**

Acute mesenteric ischemia can result from two pathologies namely embolism and thrombosis.

*Embolism*

The most common cause of AMI is superior mesenteric artery embolization. Arterial emboli are responsible for 40% to 50% of cases of AMI (19). Intra-cardiac mural thrombus is the most common source. Proximal atherogenic thoracic or abdominal aorta are potential sources. SMA arising at a less acute angle from the abdominal aorta compared with the other mesenteric vessels, makes it vulnerable to receive the emboli. The usual site of lodging of the embolus is generally distal to the origin of the middle colic artery thereby sparing the proximal few centimeters of jejunum.

*Thrombosis*

Arterial thrombosis, after embolism constitutes for the next most common cause of mesenteric ischemia accounting for 20% to 35% of

cases (20). The thrombosis can be a result of a pathology affecting the vessel wall such as atherosclerosis or those affecting the blood such as hypercoagulable state. In the case of atherosclerosis given the gradual and progressive stenosis of the vessel, patients frequently have preexisting symptoms of chronic mesenteric ischemia. Review of literature of 45 observational studies by Schoots in 2004 involving 3692 patients with acute mesenteric ischemia found that mortality from acute thrombosis of a mesenteric artery was higher compared to those suffering from acute embolism (77.4% vs. 54.1%) (21). The probable reason for this difference is attributed to the proximal location of the lesion resulting in more vessels occluded and consequently affecting larger segment of bowel.

**Non-occlusive Mesenteric Ischemia**

In the absence of a complete occlusion of the mesenteric arteries, intestinal ischemia can occur in low-velocity flow states, especially in the background of intestinal atherosclerotic disease. The most frequent primary etiology is cardiac dysfunction in the form of severe congestive heart failure and atrial fibrillation. It has to be borne in mind while in embolic occlusion mesenteric ischemia is due clot secondary to a clot in the left atrium whereas in non-occlusive mesenteric ischemia the

intestinal ischemia is secondary to reduced left ventricular output. Hepatic failure, hypovolemic shock, cardiovascular surgery, aortic incompetence and drugs are other causes (19).

**Investigations**

The investigation of the chronic mesenteric ischemia can be classifiedbroadly into the radiological demonstration of mesenteric vascular lesion and mucosal ischemia. The imaging can be further divided into non-invasive and invasive modalities. Conventional angiogram (Digital Subtraction Angiography) is the gold standard for the diagnosis of mesenteric lesion.

The sensitivity and specificity of various investigative modalities are listed below

| | Sensitivity | Specificity |
|---|---|---|
| **Abdominal arterial stenosis** | | |
| Duplex ultrasound | 88 % | 92 % |
| CTA | 82 % | 100 % |
| MRA | 100 % | 95 % |
| **Mucosal Ischemia** | | |
| Gastric Exercise Tonometry | 82 % | 87 % |
| Prolonged Tonometry | 77 % | 94 % |

**Investigations for anatomical lesion**

*Conventional Angiogram (Arteriogram)*

Catheter

The catheter type plays an important role in augmenting the angiographic image and evading injury to the vessel. They vary in shapes, sizes, and lengths. The other most important factor in deciding the choice of catheter is the site of hole at the end of catheter. It can be either end hole or multiple side holes, also called as flush catheter. Flush catheter, used in high flow large arteries, is meant for quick and uniform distribution of contrast agent at high injection pressures. While using end-hole catheters enables cannulation of remote arterial bed, injection of contrast at high pressure can cause formation of intimal flap in medium sized artery. The mode of injecting the contrast can be either hand injection or power injection.

Contrast agent

The principle of injecting contrast is to provide adequate visual contrast between the intravascular and the extravascular compartment. The timing of radiographic exposure to visualize the presence of contrast

material inside the vessel needs to be precise to avoid unnecessary exposure. Choice of the appropriate contrast agent needs to be decided taking into consideration two factors, namely, patient safety and imaging quality. Contrast agents have characteristic greater radio density than surrounding tissue making them appear darker on imaging. Agent considerably less radio dense than adjacent tissue, such as carbon dioxide ($CO_2$) gas, can also provide sufficient contrast to image intravascular anatomic detail with little compromise on the image quality (22, 23).

Conventional contrast agents are compounds with iodine being an integral part of the molecule. Iodine absorbs the x-ray photons and is responsible for the radiopacity of the contrast and thereby the visualization of the artery. Depending on dissociation of the contrast agent into anion and cation in a solution, they are categorized into ionic and non-ionic.

In ionic iodinated contrast agents, the effective osmolarity is double the original osmolarity because two ions are formed when the agent dissolves. The benzene ring in the contrast are responsible for osmolarity. The osmolarity of these agents varies from 1500 to

1700 mOsm making them ominously more hyperosmolar than plasma (285 mOsm).

Non-ionic contrast agents have considerably less osmolarity because of the aforementioned reason without affecting the radiopacity since the number of iodine atoms remain the same. Osmolarity can be further decreased by dimerization of contrast agent whereby two benzene rings are bonded to each cation resulting in formation of contrast agent with double the number of iodine atoms without increasing the osmolarity (ranging from 320 to 880 mOsm). The increased iodine atoms leads to a greater amount of x-ray photon absorption, and hence better arteriographic images.

Toxicity of iodinated contrast agent

The most common adverse effects are nausea, vomiting, and pain over the distribution of the specific vascular bed that is being studied. It is directly related to the osmolarity of the contrast agent and incidence is less with non-ionic contrast agents (24). Histamine release during contrast administration, ionic and non-ionic, is responsible for urticarial in minor cases and can cause laryngospasm at other end of spectrum

(25). Apart from the cardiac and hematological toxicity, nephrotoxicity is the most important complication.

Contrast Induced Nephrotoxicity

It ranks third among the causes of acute renal failure in hospitalized patients (26). Its presentation can vary from mild reversible rise in serum creatinine to end stage renal disease and even death. Certain population are at high risk of developing contrast induced nephropathy, the most important being pre-existing renal dysfunction (27). Other factors are increased age, severe left ventricular dysfunction, dehydration and hyperosmolar states (28). In these high risk patients, usage of minimal contrast media and pre-procedural hydration is necessary to avoid contrast induced nephropathy. However, in patents with normal renal function there seems to be little affect by the type of contrast agent and the contrast volume (29). In patients with end stage renal disease arteriogram can be performed with adequate precaution and probable need for dialysis post procedure.

Non-iodine based Contrast Agents

Other contrast agents that can be used in arteriogram are carbon dioxide and gadolinium.

Advantages of conventional angiogram

The major advantages are high sensitivity and specificity for ostio-proximal lesions and the good quality of projection of the peripheral mesenteric vasculature. The ability to delineate the pathological collateral circulation provides a road map when intervention is contemplated either endovascular or open. Real time imaging also helps in performing good quality imaging during inspiration and expiration, hence is of great importance in diagnosing celiac axis compression syndrome. The main drawbacks include the invasive nature, the potential contrast related complications and the inability to visualize the surrounding tissues. It also does not provide quantitative information about the mesenteric supply (30).

*Non-invasive imaging*

Radiography

After the advent of Computed Tomography, there is no role for plain x-ray with limited role for barium studies. The pathognomic feature of small bowel ischemia in barium studies include thickened valvulae conniventes and “thumbprinting”. There may be focal dilatation and

flattening of the mucosal folds due to the accumulation of blood in the submucosal layer.

*Doppler Ultrasound*

The diagnosis of the arterial stenosis is indirect derivative of the peak systolic flow velocity. Though the velocities are not affected in the initial stages of narrowing, peak systolic velocity of greater than 200 cm/s and 275 cm/s in celiac axis and superior mesenteric artery respectively indicate more than 70% of stenosis of these vessels. Using these criteria the detection of significant lesions in celiac axis and superior mesenteric artery is 82% and 96% respectively (11). There are patient dependent and operator dependent factors which can pose difficulty in identifying the lesions. Body habitus, dilated bowel loops and previous surgeries are improtant patient related factors. Deep location of arterial orifices, tortuous proximal celiac axis make it less accessible for sonographic studies. Inappropriate placement of probe can lead to erroneous doppler angle and spectral waveforms. Despite these, given its high diagnostic accuracy in significant lesions when performed by experts, doppler study can be a useful screening tool in high risk individuals and initial investigation in patients with suspected chronic mesenteric ischemia rather than invasive angiogram.

*Multidetector Computed Tomography Angiogram and Magnetic Resonance Angiogram*

By combining the virtue of non-invasiveness and advantages of conventional angiogram, computed tomography angiogram is being preferred in the evaluation of patients with acute and chronic mesenteric ischemia. In addition to the information provided by the conventional angiogram, CT detects tiny distal vascular segments and most importantly can characterise the plaque lesion identifying unstable plaques. The imaging of the fat, mesentery and omentum can help in identifying infarction. Magnetic resonance imaging is comparable to CT angiogram but cannot be performed in acute setting.

**Investigations for mucosal ischemia**

There is a necessity to prove intestinal ischemia even if the study of mesenteric vessels shows narrowing especially in single vessel disease. This not only confirms the diagnosis of mesenteric ischemia but also determines the outcome when intervention is planned. Even in a single vessel disease with greater than 50% stenosis presenting with abdominal symptoms, 60% truly have mucosal ischemia and will benefit from revascularizaion. Gastrointestinal tonometry is an established technique

to detect the mucosal ischemia. It involves the estimation of gastric mucosal carbon dioxide. In states of tissue hypoperfusion, acute shock or in chronic insufficiency, the carbon dioxide produced by the tissue during metabolism is not washed by the blood resulting in its accumulation. This is a global phenomenon and the stomach being accessible organ by placement of nasogastric tube, gastric tonometry can be indirect evidence of perfusion to the rest of the gastrointestinal tract. Mensink during evaluation of 354 patients found the tonometry to have a sensitivity and specificity of 78-85% and 82-92% respectively in the detection of mesenteric ischemia (31).

Based on the principle of inducible ischemia in cardiac patients similar tests have been evolved based on exercise induced and meal provoked. Even with postprandial tonometry, 4 out of 17 patients with mesenteric ischemia were missed (32). The reason for this is attributed to the fact that mucosal ischemia can last for short periods and entirely reversible in the intervening periods.

Gastrointestinal tonometry combined with duplex ultrasound has been recommended as the best diagnostic strategy for individuals with suspected chronic mesenteric ischemia (33). Otte et al found screening with this combinedmodality would identify patients with symptomatic

ischemia and at the same time would have avoided 21% of unnecessary angiographies.

Recent advances include visible light spectroscopy during endoscopy to ascertain tissue saturation and probable serum markers (34, 35). If these tests can be validated, they can provide easy accessibilty and may lead to a considerable increase in the diagnosis of chronic mesenteric ischemia.

**Coronary Artery Disease**

The importance of the disease process and its pandemic nature was probably not thought of when the first issue of the well renowned journal New England Journal Medicine and Surgery carried an article titled “Remarks on Angina Pectoris” by John Warren (36). As with most medical conditions, the treatment of the disease was based on assumptions rather than scientific basis. The situation though has improved but by no means has the problem of atherosclerotic coronary artery disease is nearing complete solution.

With no investigations, there was significant delay in identifying the fact that occlusion of coronary artery by thrombosis results in the clinical features of angina pectoris and eventually the pathological

occurrence of myocardial infarction. This is evident by the fact that clinical description of angina by Heberden was in 1772; but the pathological identification of coronary thrombosis resulting in myocardial ischemia was described a century later by Ludvig Hektoen in 1879 (37, 38). Following years saw better understanding between clinicians and pathologist trying to explain the concept of myocardial infarction. The first major breakthrough in identifying the disease process in living individual was done by Herrick when he used electrocardiography in five patients to diagnose myocardial infarction (39).

With the increasing use of electrocardiogram and awareness on symptomatology of coronary artery disease, the focus shifted from identifying the onset of myocardial infarction to identifying risk factors of coronary artery disease. This was primarily driven by the fact that people belonging to the reproductive age group suffered a lot. The first well known study, based on the assessing the risk factors prevalent in the residents of Framingham, put forth that hypertension and elevated cholesterol levels were common in patients with ischemic heart disease and acute myocardial infarction. This finding formed an important link

in connecting the basic pathology of atherosclerosis and myocardial infarction

**Anatomy and Physiology of Coronary blood flow**

The heart is supplied by a group of epicardial vessels which are direct branches of aorta. They arise from the aortic root with the origin in relation to the sinus of Valsalva. Though there are three aortic sinuses, namely anterior, left posterior, and right posterior, there are only two arteries commonly present, left and right coronary artery.

*Left Coronary Artery*

The left coronary artery originates from the left coronary sinus in relation to the left cusp of the aortic valve. The common trunk called as left main artery has a very short course and divides to form the left anterior descending artery and the left coronary circumflex artery.

*Left Anterior Descending Artery (LAD)*

It courses in the interventricular sulcus from the aortic root toward cardiac apex. It generally crosses the acute margin of the heart, margin dividing the anterior surface of the heart from the inferior surface of the heart. At the end of the interventricular groove in the diaphragmatic

surface it forms an anastomoses with right system of the heart through posterior descending coronary artery. Throughout its course the artery gives off multiple perforating branches into the anterior part of the IVS. The first of these branches carries immense clinical importance in that it supplies the region of the bifurcation of the common atrioventricular bundle.

Apart from these perforators the LAD typically provides several branches, which travel in an oblique course towards the left side and apex. It is for this reason they are aptly called as diagonal branches. They pass parallel to the superficial layer of myocardium in contrast to the perforators which pass perpendicular to the myocardium.

*Left Circumflex Artery (LCX)*

The left-circumflex coronary artery (LCX) courses in the left atrio-ventricular groove to reach the posterior surface of left ventricle. During its course the left circumflex gives off obtuse marginal branches (OM). These branches predominately supply the anterior, anterolateral and the posterior surface of left ventricle.

The termination of the left circumflex artery is variable. And the type is classified as dominance. When the left coronary circumflex artery gives

origin to the posterior descending artery and the atrioventricular node artery, the pattern of coronary artery distribution is classified as left coronary dominance. This more commonly seen in males.

It should be borne in mind that the dominance is not decided by the blood supply of the entire interventricular septum but only the posterior part of the interventricular septum since the anterior 2/3$^{rds}$of the interventricular septum is invariably supplied by the left anterior descending artery.

The type of coronary distribution where the right coronary artery gives of the posterior descending artery is called as right dominance. The clinical relevance of this dominance is due to the presence of anastomotic interconnections between the left and the right system of the coronary circulation and thereby providing a possible collateral coronary supply in the event of ischemic heart disease affecting any one of the coronary system.

*Right Coronary Artery*

It originates from the right coronary sinus of Valsalva. Its blood supply distribution usually includes the entire right atrium and the right ventricle. Right coronary artery is said to be “dominant” when the

posterior descending artery arises from the right coronary artery. During this circumstance, a part of the posterior left atrium and left ventricle, the posterior papillary muscle of the LV, and the A-V node also derive their blood supply from the right system.

The conus artery is the first noteworthy branch of the right coronary artery. It frequently passes in anti-clockwise direction around the infundibulum. Arterial circle of Vieussens can be present when the conus artery anastomoses with a branch of the left anterior descending artery. It has also been found that in the elderly, the conus artery can arise from a separate aortic ostium. This variant is attributed to the growth of the aortic sinus, where the origin of the conus artery comes to be progressively integrated into the aortic valve. The knowledge of these variants is essential during surgical and endovascular intervention.

The most common of these variants is an anomalous beginning of the main left or right coronary artery from the erroneous sinus of Valsalva (40).

*Sinoatrial Artery*

The next branch of the right coronary artery ascends along the anterior wall of the RA regularly but arises from the deeper side of the

RCA. It supplies the sinoatrial node. This occurs in about 40 percent of individuals

Several branches supplying the anterior right ventricle are given off as the artery courses along the right atrioventricular groove. As the RCA crosses the margin to reach the diaphragmatic surface a right marginal branch called arteria margo acutus is given off. The right coronary artery coursing in the posterior right atrioventricular groove, near the crux, gives origin to posterior descending artery, atrioventricular nodal artery and a branch to the inferior surface of the left ventricle.

**Coronary Distribution and Anastomoses**

The epicardial arteries which course beneath the epicardium gives rise to the perforating branches which enter the myocardium. These perforators in turn give rise to small branches which arborize instantly to supply the outer two-thirds of the myocardium. The perforating arteries carry on, devoid ofadditional branching, to end in a large subendocardial plexus.

Homocoronary, within the same system, and intercoronary anastomoses between the right and left system can be of pronounced significance in cardiac function and coronary artery disease.

Intercoronary anastomoses that commonly present are:

- At the origin of the pulmonary conus, between the LAD and the conus branch of the RCA
- At the interventricular groove, between the anterior (LAD) and posterior descending arteries (PDA)
- On the diaphragmatic surface of the left ventricle, amongst terminal branches of the right coronary artery and the LCX
- Within the substance of the interventricular septum, between perforating septal branches of the anterior and posterior descending arteries

**Coronary artery disease**

The spectrum of clinical presentation of coronary artery disease varies from asymptomatic individuals to sudden cardiac arrest (41).While the two extremes are difficult to detect or treat, it is the intervening phase of the disease process which forms the crucial period of effective intervention. The demand supply mismatch during episodes of ischemia causes momentary derangement of the mechanical, biochemical, and electrical functions of the myocardium. The tendency for coronary

atherosclerosis to be focal results in asymmetrical ischemia. Myocardial pump function can be altered due to ventricular contractility disturbances which might be in the form hypokinesia, dyskinesia or akinesia.

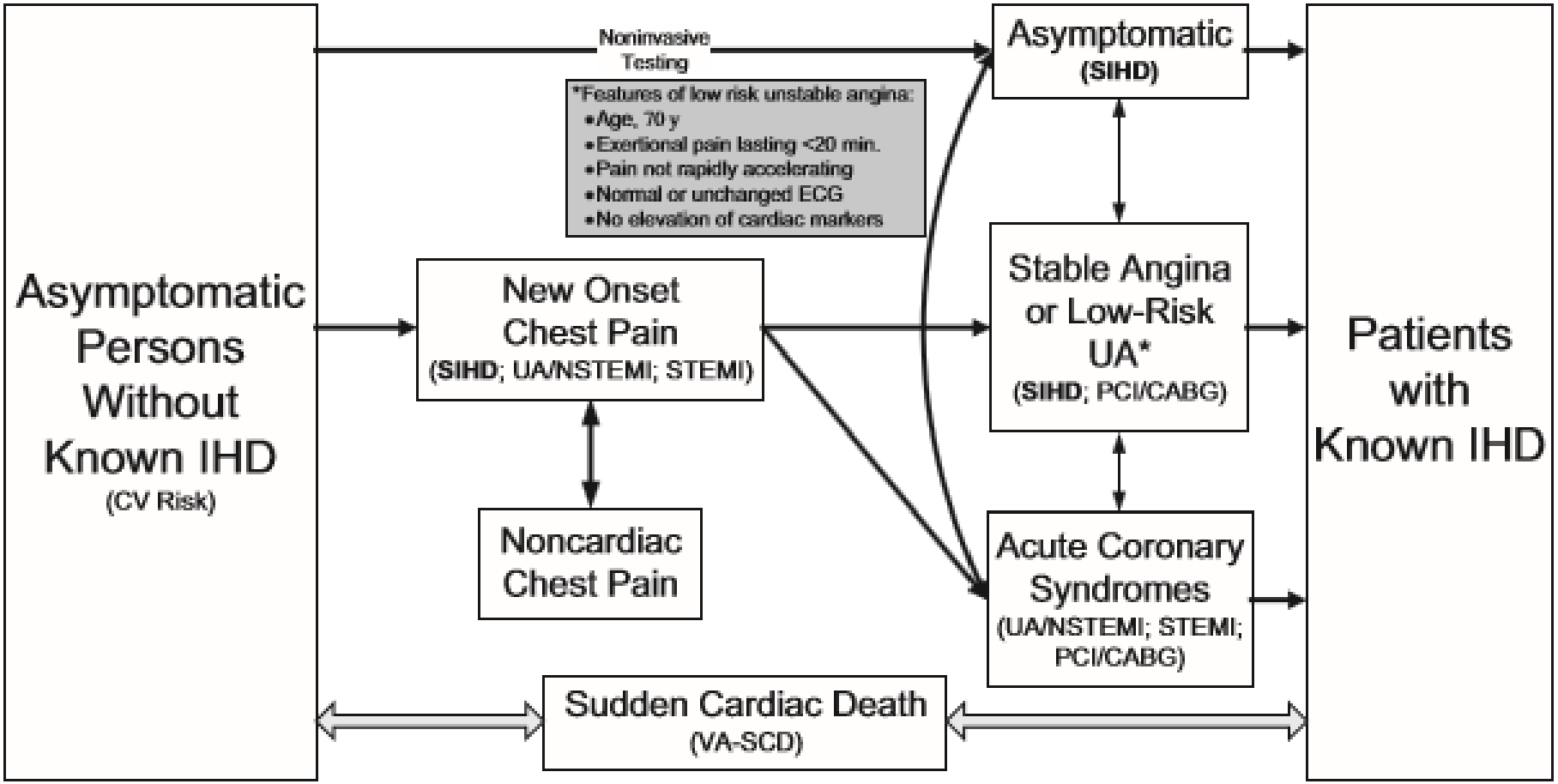
While the gradual occlusion of the coronary vessels is associated with episodes of ischemia, an acute subtotal or total occlusion of coronary vessel is accompanied by instant failure of myocardial activity. There is a differential level of ischemia within the cardiac layers with the subendocardium being most affected. The involvement of the ventricular myocardium causes transitory left ventricular failure. Valve dysfunction can occur if the papillary muscles are involved in ischemia. The duration of insufficiency becomes important. While transient ischemia causes angina pectoris, prolonged periods can result in myocardial necrosis and scarring.

Anginal pain is characterized by crescendo-decrescendo pattern lasting less than 5 minutes and radiating to either shoulder or arms. It is usually precipitated by exertion or emotional disturbance. Exertional angina is typically relieved by rest and sublingual nitroglycerin. The physical examination is unremarkable in patients with stable angina during the angina-free period. The patients can have stigmata of atherosclerosis

like xanthelasmas or nicotine staining indicating smoking habit. Barring C-reactive protein, laboratory investigations are not of much value in ischemic heart disease.

12-lead electrocardiogram is the initial investigation of choice though it may be normal when recorded at rest in patients with typical angina pectoris. Though changes in electrocardiogram can suggest ischemic heart disease they are non-specific. The changes that can accompany ischemic heart disease are repolarization abnormalities, interventricular conduction defects and cardiac rhythm disturbance. Electrocardiogram changes suggestive of old myocardial infarction definitely indicate an underlying coronary artery disease.

Echocardiography evaluates the left ventricular ejection fraction in patients with coronary artery disease. Affectation of ventricular myocardium results in both global and regional wall motion abnormalities of the left ventricle. In latent cases electrocardiogram and echocardiogram done before and after exercise can make evident the signs of ischemia.


Asymptomatic Persons Without Known IHD
(CV Risk)
Noninvasive Testing
*Features of low risk unstable angina:
•Age, 70 y
•Exertional pain lasting <20 min.
•Pain not rapidly accelerating
•Normal or unchanged ECG
•No elevation of cardiac markers
New Onset Chest Pain
(SIHD; UA/NSTEMI; STEMI)
Noncardiac Chest Pain
Asymptomatic
(SIHD)
Stable Angina or Low-Risk UA*
(SIHD; PCI/CABG)
Acute Coronary Syndromes
(UA/NSTEMI; STEMI; PCI/CABG)
Sudden Cardiac Death
(VA-SCD)
Patients with Known IHD

## Management of coronary artery disease

The initial line of management is lifestyle modification and pharmacotherapy which are combined together to treat the various cardiovascular risk factors such as hypertension, hyperglycemia, dyslipidemia and obesity. Failure of these approaches, intervention is required, percutaneous or surgery, which are based on identification and removal of the stenosis(42).

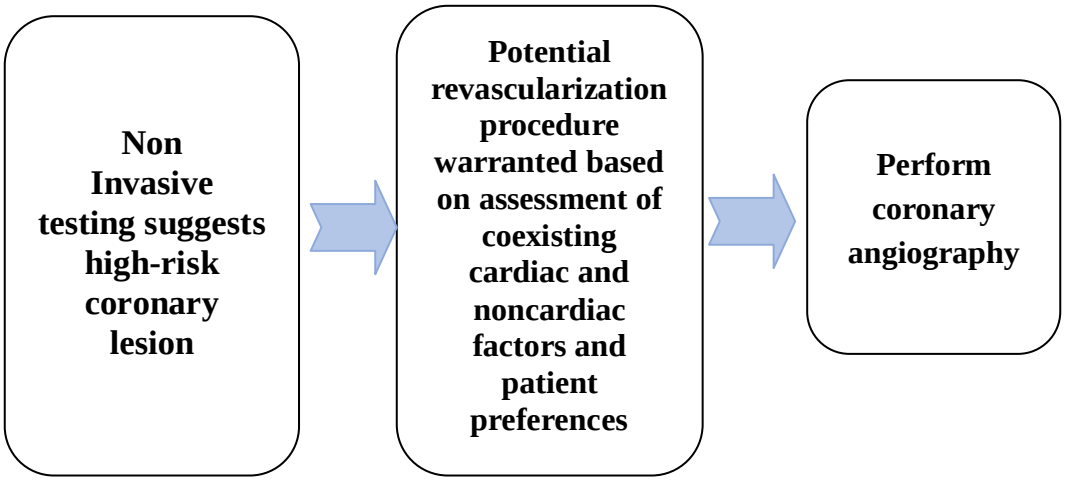
## Coronary Angiogram

Coronary angiogram demonstrates the lumen of the coronary arteries and hence detect coronary artery stenosis or occlusion. The test however is not suitable for evaluating the arterial wall and characterize the plaque if present. This gives way for false negative results when atherosclerotic lesion that does not intrude on the lumen may go hidden.

*Indications of coronary angiography*

Patients with Stable Ischemic Heart Disease, who have survived sudden cardiac death or potentially life-threatening ventricular arrhythmia should undergo coronary angiography to assess cardiac risk (43).

Patients with SIHD who develop symptoms and signs of heart failure should be evaluated to determine whether coronary angiography should be performed for risk assessment(44).



# REVIEW OF LITERATURE

## Pathology of atherosclerosis

The pathological process of atherosclerosis is a multifactorial entity predominately affecting the large and medium- sized arteries. The process which is now believed to start from infancy gradually increases, eventually compromising the vascular lumen. This vascular insufficiency leads to the dysfunction of the organs whose blood supply is compromised (45). The location of atherosclerotic plaques in the mesenteric circulation is characteristically at the ostium. These lesions are thought to be primary extensions of progressive atherosclerosis of the anterior aortic wall (15).

Chronic mesenteric ischemia is a disorder posing diagnostic challenge and often the recognition is delayed till the setting of acute on chronic mesenteric ischemia which is associated with high mortality and morbidity. The delay in identification of the disorder by treating physician and the need for confirmation of the disorder by an invasive test, angiogram, form an important component of the eventual delay (33). With the advent of non-invasive screening tests like duplex scanning, serum markers and visible light spectroscopy, the diagnosis of

chronic gastrointestinal ischemia can be made at an earlier stage. Since bowels need not suffer from ischemia even in the presence of stenosis, demonstration of the mucosal ischemia is necessary for the diagnosis of mesenteric ischemia. Gastrointestinal tonometry is probably the only modality that demonstrates physiological changes that accompany actual ischemia. It involves the measurement of luminal $PCO_2$ in comparison to arterial $PCO_2$ and can detect early stages of vascular insufficiency.

**Poly-vascular atherosclerotic disease**

Though the presence of the global process should affect the vascular beds throughout the body, there is heterogeneity in their occurrence. This is hypothesized to be due to differential risk factor profiles for the various predilection places (45).This has grave implications on the management of these vascular diseases, since failure to identify and treat a co-existing vascular lesion at another site might significantly alter the prognosis.

The medical management of atherosclerotic vascular disease is essentially the same involving cessation of smoking, lifestyle

modification, statins, antiplatelet agents, glycemic and blood pressure control.

The largest study, to date, directed to identify coexisting lesions in known coronary artery disease patients is from Imori et al in January 2013 from Japan. The study included 1734 patients who underwent diagnostic angiogram for suspicious coronary artery disease between September 2010 and July 2011. The carotid artery and the renal artery involvement were identified by means internal carotid artery duplex scanning and renal artery Doppler study respectively. The involvement of the peripheral arterial disease in the lower limb was determined by ankle-brachial index measurement using continuous wave Doppler probe. 1253 patients were confirmed to be having coronary artery disease. 24% of the studied population had more than one site involved with atherosclerotic lesion thus vindicating the rationale behind the study. The prevalence of carotid artery, renal artery and lower limb peripheral artery in patients with coronary artery disease was found to be 7%, 9% and 16% respectively. Involvement of any two sites was found in 7% and all three sites in 0.8% of studied population. The study put forth various independent risk factor for each vascular bed among the common cardiovascular risk factor. With respect to coronary artery

disease, single vessel disease was not significantly associated with atherosclerotic lesions at other site. The presence of left main disease was found to be an independent risk factor for carotid artery, renal artery and lower extremity peripheral artery (46).

**Chronic mesenteric Ischemia in known atherosclerosis**

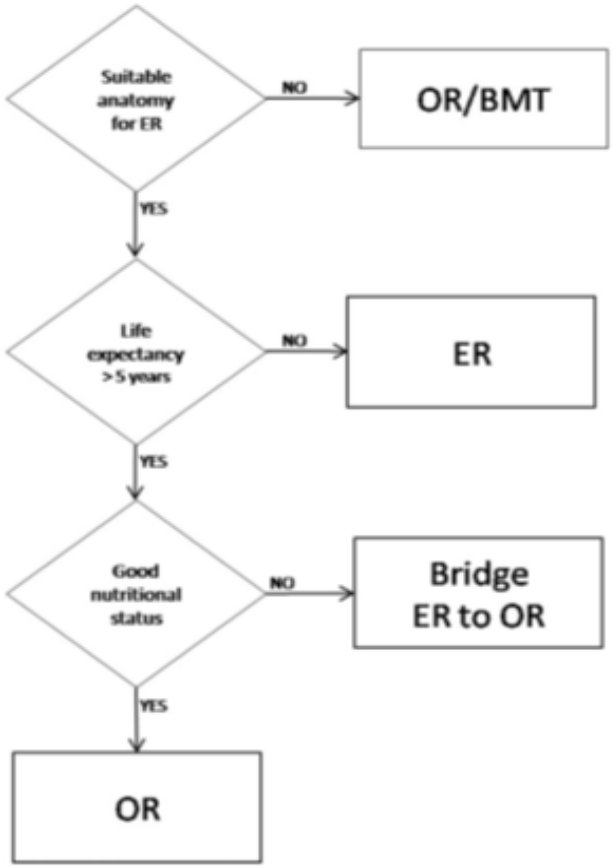
Chronic gastrointestinal ischemia is another extra-cardiac manifestation of atherosclerosis. Very few studies have compared the prevalence of mesenteric vascular diseases and the relationship with classical cardiovascular risk factors. Consensus has not been arrived at, since these studies are plagued by differential age distribution and small study population. Another important drawback in majority of these western based studies is that they were conducted in patients who underwent revascularization which may not be a true reflection of the mesenteric vascular disease.

After an ischemic episode event at any site, there is high absolute risk for a recurrent ischemic event in the same or in any other vascular bed (47). Similarly, poly-vascular disease patients on comparison with mono-vascular disease patients have a doubled risk of cardiovascular related mortality (48).

In an analysis of 195 patients, atherosclerotic chronic gastrointestinal ischemia belonged to upper age group and were more often smokers with positive family history. They suffered from profound weight loss when compared to the control group (49).

**Need for revascularization**

Indication for revascularization has undergone dramatic changes in the last two decades. Generally fraught with high morbidity and mortality in the past, the open surgical revascularization, is being gradually being replaced by endovascular approach as the primary modality of revascularization. The high prevalence of the atherosclerotic lesion to involve the ostio-proximal segment of the mesenteric arteries and short segment involvement makes them accessible through endovascular approach (50). Revascularization is mandatory once the patients develop symptoms in order to prevent bowel infarction (51). The factors that are crucial in the decision making regarding revascularization include suitable anatomy, life expectancy and nutritional status. Apart from these, if procedures are planned for aneurysmal disease of the aorta and in presence of total stump occlusion, then open procedures are the treatment of choice (52). The following provides a useful algorithm for decision making regarding the type of revascularization.




The controversy lies in the treatment of asymptomatic individual. Though it was considered that intervention in asymptomatic individual is not warranted, better understanding of the natural history of the chronic mesenteric ischemia has seen changing concepts in this field. The rationale for this concept stems from the fact that nearly 50 % of mesenteric ischemia patients have acute infarction as their first manifestation (53, 54).

Screening of asymptomatic elderly (>65 years) Americans in 2006, based on Doppler flow ultrasound-derived criteria showed that 17.5% of

the participants had critical mesenteric artery stenosis. On follow-up for more than 6 years none of them developed symptoms or intestinal infarction related mortality (55). In contrast, an arteriography based study by Thomson et al in 1998 found 82 of 980 asymptomatic individuals having at least one artery stenosis by more than 50%. They reported that 4 of 15 patients with significant stenosis affecting all three arteries developed mesenteric ischemia during a mean follow-up of 2.6 years (53). 13 out of 15 patients with triple vessel disease developed abdominal pain or died during six year follow-up. Thus revascularization has to be considered in patients with known cardiovascular risk factors with significant triple-vessel disease even if the patient is asymptomatic. The fact that 15% to 50% of individuals presenting with acute mesenteric ischemia do not have any antecedent warning signs underlines the significance of early intervention (56). Hence asymptomatic individuals with significant stenosis of mesenteric vessels are candidates for endovascular intervention.

Though revascularization procedures have evolved, there is still significant morbidity and mortality associated these procedures and these have to be carefully weighed against risk of mesenteric ischemia in asymptomatic individuals.

# AIM & OBJECTIVES

**Aim**

To study the prevalence of mesenteric vascular disease in ischemic heart disease patients

**Objectives:**

To assess the symptomatology of mesenteric vascular disease

To assess risk factors for atherosclerotic mesenteric vessel disease

# MATERIALS AND METHODOLOGY

Place of study:

Govt. Stanley Medical College and Hospital, Chennai

Duration:

January 2013 to June 2013

Study design:

Prospective observational study

Inclusion criteria:

Patients with suspected ischemic heart disease admitted for coronary angiogram

Exclusion criteria:

1. Patients with normal coronaries on angiogram
2. Patients with elevated renal parameters

Methodology:

Study of 110 consecutive patients with suspected ischemic heart disease admitted for coronary angiogram in the Department of Cardiology from January 2013 to June 2013. After obtaining informed consent, patients were enrolled for the study.

A detailed history taking, with emphasis on the risk factors of atherosclerosis and abdominal symptoms was done followed by complete clinical examination. Allen test was performed to confirm the patency of the palmar arches.

Patient was subjected to basic investigation and if renal parameters were normal, then patient was subjected to angiogram. Echocardiogram was performed as a part of cardiac evaluation.

Patient preparation included administering Inj. Tetanus Toxoid if indicated, Inj. Lignocaine test dose and preparation of both wrist and groin.

Procedure

- Pre-procedure hydration maintained by administration of intravenous fluids

- Right radial artery was the preferred site of catheterization.
- Local anesthesia administered at the local site.
- Catheterization was done by Seldinger technique.
- To prevent catheter clot formation - fluid containing Inj. Heparin 500 IU diluted in 1 litre of NS was used for flushing
- Coronary angiogram was performed
- If coronary arteries were involved, then mesenteric angiogram was done
- Selective cannulation of the three mesenteric arteries was done, and angiogram performed in two views - antero-posterior and lateral views
- After the procedure, compressive dressing was applied
- During the procedure, patient ECG, pressure and oxygen saturation was continuously monitored.
- Patient was watched for anaphylactic reaction and contrast induced nephrotoxicity
- Generally patient was discharged after 24 hours

Patients were followed up for 3 months with symptoms.

If patient had lesions significant enough to warrant revascularization, they were advised to undergo intervention. (Only one patient underwent endovascular revascularization).

# TABLES

**Table 1 Demographic Characteristics of study population**

| | **All (n=103)** | **Mesenteric vascular disease (n=44)** | **No Mesenteric vascular disease** |
|---|---|---|---|
| *Age* | 56.31 | 60.63 | 53.08 |
| *Gender (M/F)* | 66/37 | 24/20 | 42/17 |
| *Abdominal Symptom (Y/N)* | 26/77 | 19/25 | 7/52 |
| *Diabetes Mellitus (Y/N)* | 41/62 | 29/15 | 12/47 |
| *Hypertension (Y/N)* | 23/80 | 10/34 | 13/46 |
| *Known Ischemic heart Disease (Y/N)* | 23/80 | 13/31 | 10/49 |
| *Peripheral Arterial Disease (Y/N)* | 21/82 | 19/25 | 2/57 |
| *Dyslipidemia (Y/N)* | 32/71 | 15/29 | 17/42 |
| *Smoking (Y/N)* | 40/63 | 20/24 | 20/39 |
| *Alcohol (Y/N)* | 33/70 | 14/30 | 19/40 |
| *BMI (Underweight/Normal/Overweight)* | 8/46/49 | 6/29/9 | 2/17/40 |

**Table 2 Mesentery artery-wise involvement**

| ***Mesenteric artery*** | **Involvement** |
|---|---|
| *Celiac* | 22 |
| *SMA* | 39 |
| *IMA* | 15 |

**Table 3 Coronary artery-wise involvement**

| ***Coronary Artery*** | **Involvement** |
|---|---|
| *RCA* | 52 |
| *LAD* | 76 |
| *LCX* | 63 |

**Table 4 Need for Revascularization in mesenteric arterial involvement**

| | | **Mesenteric artery involvement** | **P value** |
|---|---|---|---|
| *Need for Revascularization* | Yes | 18 | <0.01 |
| | No | 26 | |
| *Need for Open Revascularization* | Yes | 12 | 0.5 |
| | No | 32 | |

Significance at $P < 0.05$. Chi-Square test

**Table 5 Mesenteric vessel involvement with epidemiological factors**

| *Parameter* | Classification | Patients with Mesenteric artery involvement | P- Value |
|---|---|---|---|
| *Age* | < 65 years | 19 | **< 0.01** |
| | ≥ 65 years | 25 | |
| *Gender* | Male | 24 | 0.08 |
| | Female | 20 | |
| *Symptomatic* | Yes | 19 | **<0.01** |
| | No | 25 | |
| *Smoking* | Yes | 20 | 0.3 |
| | No | 24 | |
| *Alcohol* | Yes | 14 | 0.9 |
| | No | 30 | |

Significance at P < 0.05. Chi-Square test

**Table 6 Significant clinico-biochemical parameters in mesenteric artery involvement**

| *Parameter* | Classification | Patients with Mesenteric artery involvement | P- Value |
|---|---|---|---|
| ***BMI*** | < 25 | 35 | **< 0.01** |
| | ≥ 25 | 9 | |
| ***Serum albumin*** | < 3.5 | 20 | **< 0.01** |
| | ≥ 3.5 | 24 | |
| ***ESR*** | Elevated | 27 | **<0.01** |
| | Normal | 17 | |

Significance at P < 0.05. Chi-Square test

**Table 7 Co – morbidities with mesenteric artery involvement**

| ***Co – Morbid Disease*** | **N** | **Odds Ratio** | **95% CI** |
|---|---|---|---|
| *Diabetes Mellitus* | 29 | **7.57** | **3.11 - 18.42** |
| | 15 | | |
| *Hypertension* | 10 | 1.04 | 0.40 - 2.65 |
| | 34 | | |
| *Known Ischemic heart disease* | 13 | **2.05** | **0.80 - 5.25** |
| | 31 | | |
| *Peripheral Vascular Disease* | 19 | **21.66** | **4.68 - 100.13** |
| | 25 | | |
| *Dyslipidemia* | 15 | 1.27 | 0.51 - 2.96 |
| | 29 | | |

**Table 8 Co - Morbidities with need for revascularization**

| ***Co – Morbid Disease*** | **N** | **Odds Ratio** | **95% CI** |
|---|---|---|---|
| *Diabetes Mellitus* | 11 | **2.47** | **0.89 - 6.82** |
| | 8 | | |
| *Hypertension* | 4 | 0.91 | 0.27 - 3.07 |
| | 15 | | |
| *Known Ischemic heart disease* | 9 | **4.50** | **1.54 - 13.09** |
| | 10 | | |
| *Peripheral Vascular Disease* | 8 | **3.97** | **1.34 - 11.76** |
| | 11 | | |
| *Dyslipidemia* | 5 | 0.75 | 0.24 - 2.30 |
| | 14 | | |

**Table 9 Presence of Mesenteric artery disease with number of coronary artery involvement**

| *Coronary Artery involved* | Mesenteric arterial disease | | P value |
|---|---|---|---|
| | Yes | No | |
| *One* | 12 | 25 | 0.13 |
| *Two* | 19 | 25 | |
| *Three* | 13 | 9 | |

Significance at P < 0.05. Chi-Square test

**Table 10 Specific coronary artery with mesenteric artery disease**

| *Coronary Artery* | Frequency | Mesenteric arterial disease | | P value |
|---|---|---|---|---|
| | | Yes | No | |
| *RCA* | 52 | 22 | 30 | 0.9 |
| *LAD* | 76 | 37 | 39 | 0.04 |
| *LCX* | 63 | 30 | 33 | 0.2 |

Significance at P < 0.05. Chi-Square test

**Table 11 Specific Coronary Artery with need for revascularization**

| *Coronary Artery* | Involvement | Need for Intervention | | P- Value |
|---|---|---|---|---|
| *RCA* | 52 | 11 | 41 | 0.61 |
| *LAD* | 76 | 16 | 60 | 0.38 |
| *LCX* | 63 | 15 | 48 | 0.11 |

Significance at P < 0.05. Chi-Square test

**Table 12 Specific mesenteric artery with need for revascularization**

| ***Mesenteric artery*** | **Involvement** | **Need for revascularization** | | **P- Value** |
|---|---|---|---|---|
| | | Yes | No | |
| *Celiac* | 22 | 10 | 12 | **< 0.01** |
| *SMA* | 39 | 15 | 24 | **< 0.01** |
| *IMA* | 15 | 7 | 8 | **< 0.01** |

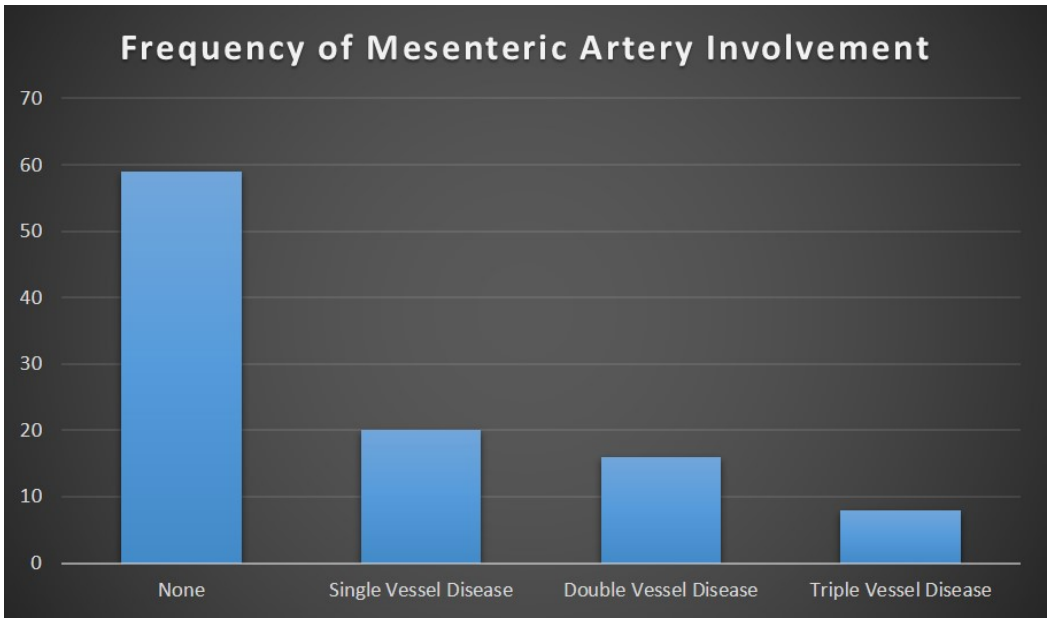
Significance at P < 0.05. Chi-Square test

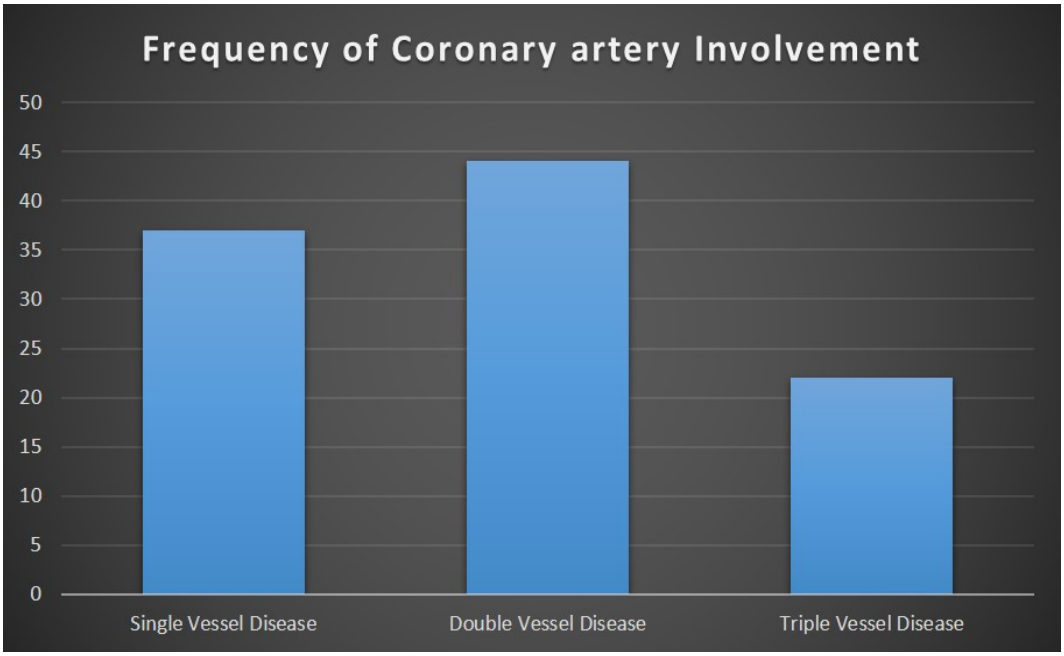
**Table 13 Specific Mesenteric artery with need for surgical intervention**

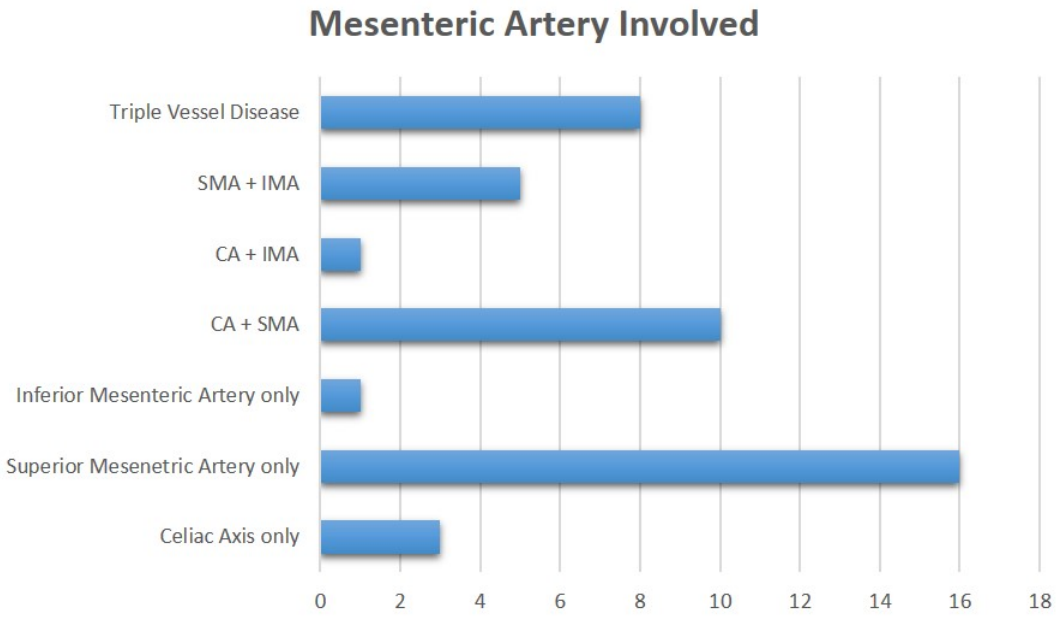
| ***Mesenteric artery*** | **Involvement** | **Need for Surgical Intervention** | | **P- Value** |
|---|---|---|---|---|
| *Celiac* | 22 | 9 | 13 | **< 0.01** |
| *SMA* | 39 | 11 | 28 | **< 0.01** |
| *IMA* | 15 | 7 | 17 | **< 0.01** |

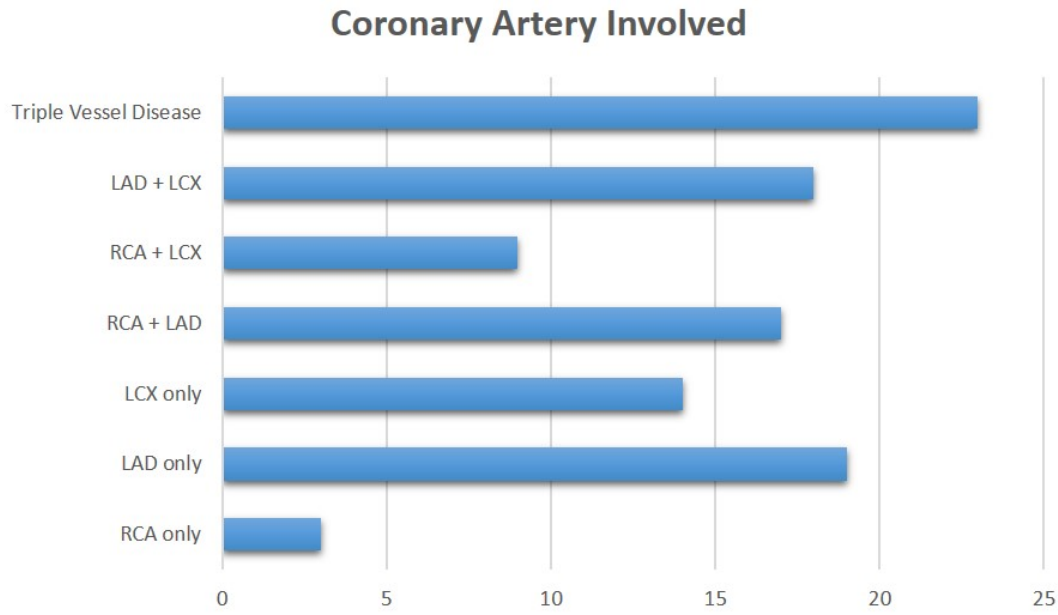
Significance at P < 0.05. Chi-Square test

# CHARTS


Frequency of Mesenteric Artery Involvement
70
60
50
40
30
20
10
0
None
Single Vessel Disease
Double Vessel Disease
Triple Vessel Disease


Frequency of Coronary artery Involvement
50
45
40
35
30
25
20
15
10
5
0
Single Vessel Disease
Double Vessel Disease
Triple Vessel Disease


Mesenteric Artery Involved
Triple Vessel Disease
SMA + IMA
CA + IMA
CA + SMA
Inferior Mesenteric Artery only
Superior Mesenetric Artery only
Celiac Axis only
0
2
4
6
8
10
12
14
16
18


Coronary Artery Involved
Triple Vessel Disease
LAD + LCX
RCA + LCX
RCA + LAD
LCX only
LAD only
RCA only
0
5
10
15
20
25

# IMAGES

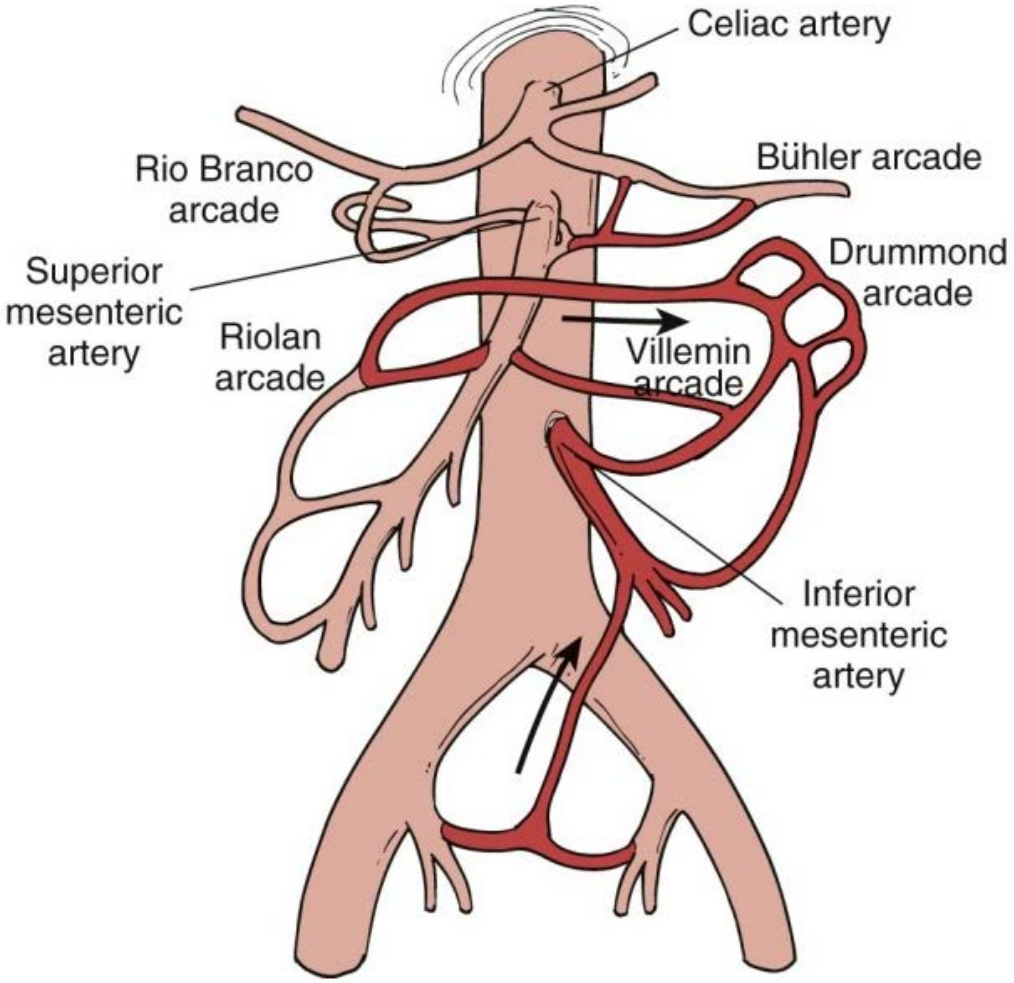



**Figure 1 Collateral network in Mesenteric blood supply**

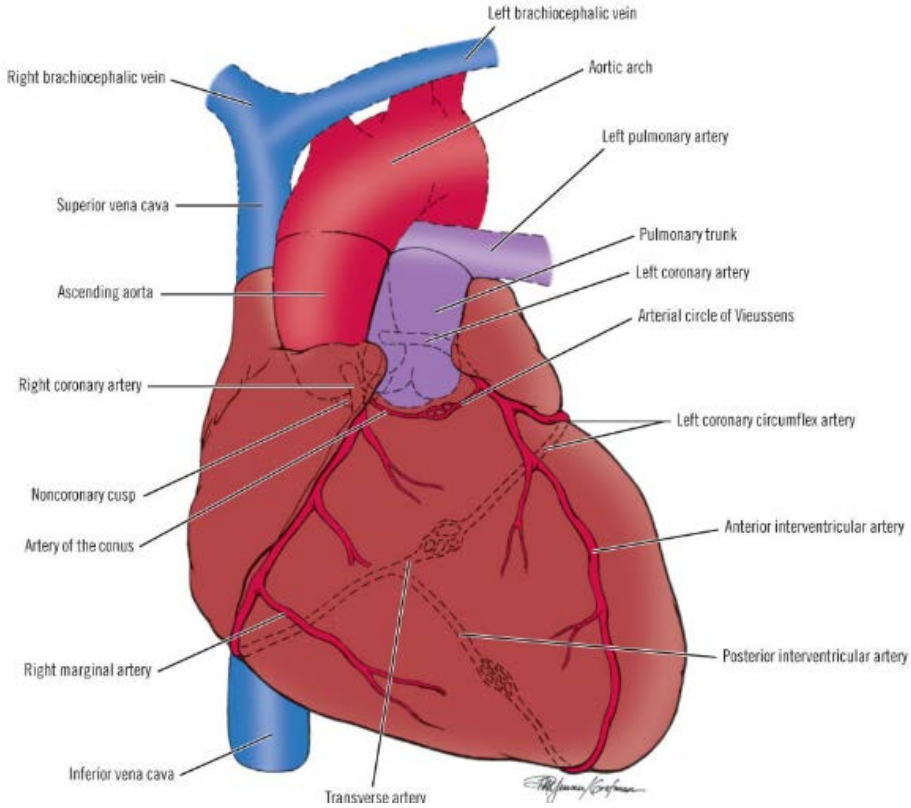



**Figure 2 Blood Supply of the heart**

**Figure 3 Dr. Sven Ivar Seldinger (1921–1998)**

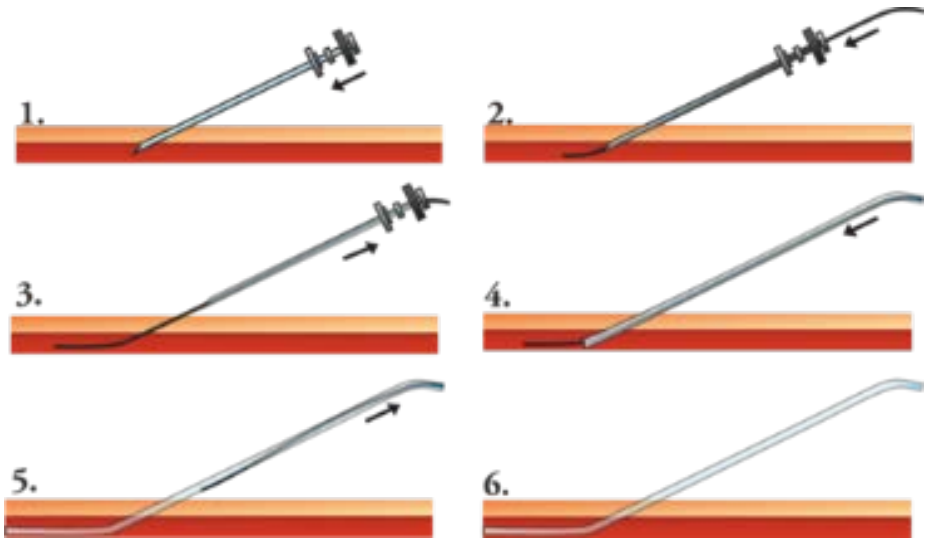



**Figure 4 Seldinger technique 1. Insertion of needle. 2. Introduction of guidewire. 3. Removal of needle. 4. Advancement of dilator over the guidewire. 5. Advancement of catheter over guidewire. 6. Removal of guidewire.**

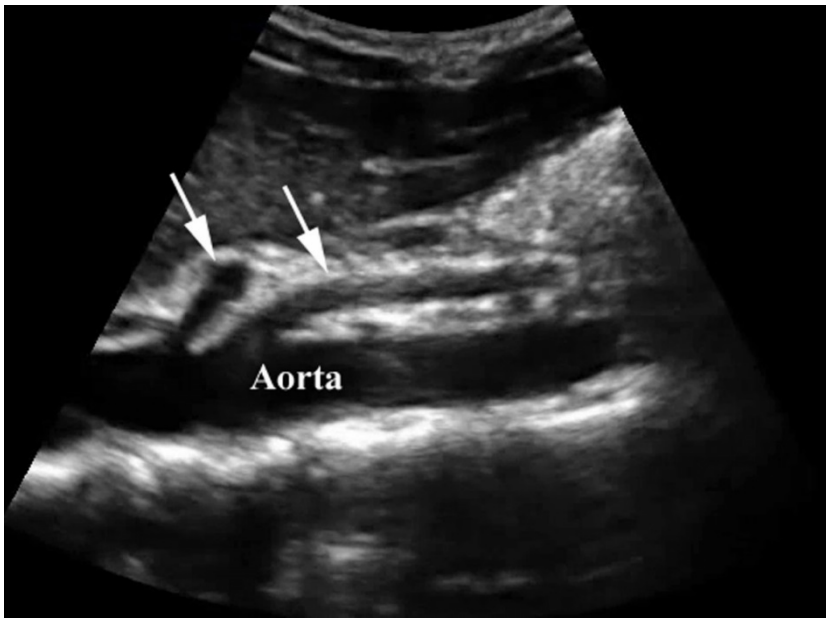



**Figure 5 B-Mode study of Mesenteric Vessels**

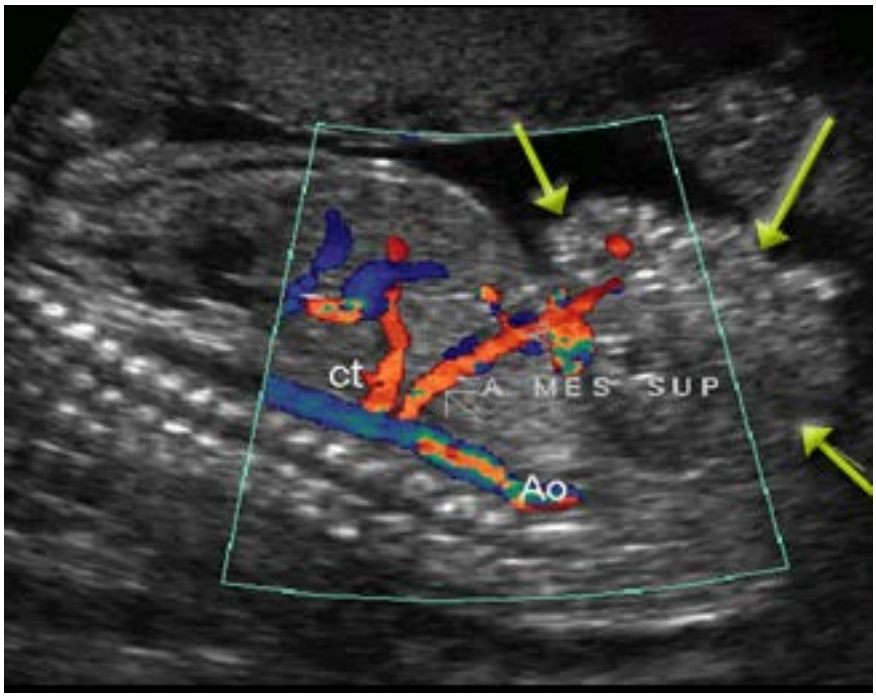



**Figure 6 Doppler study of Mesenteric vessels**

**Figure 7 Computed Tomography of Superior Mesenteric Artery (SMA) - MIP view**

**Figure 8  Computed Tomography of Superior Mesenteric Artery (SMA)-VR view**

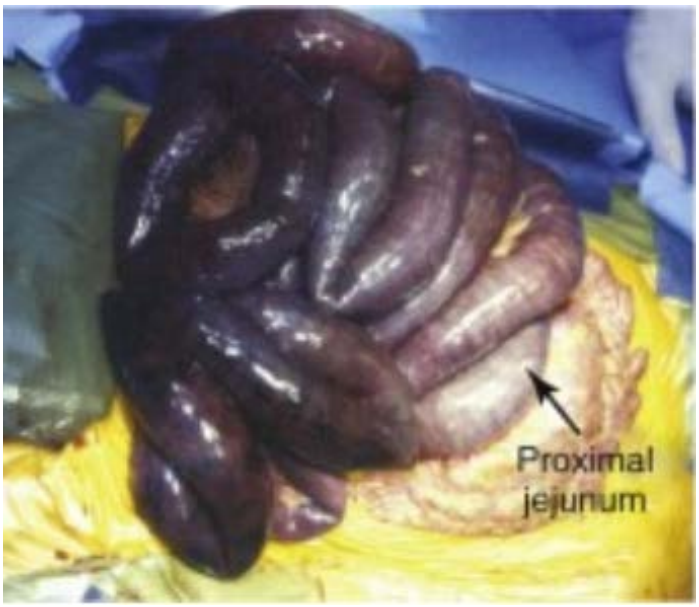



**Figure 9 Acute Embolic Mesenteric ischemia - the proximal jejunum is spared**

**Figure 10 Acute thrombotic mesenteric ischemia**

**Figure 11 Characteristic clinical feature of patient with chronic mesenteric ischemia**

**Figure 12 Pneumatosis Intestinalis in CT abdomen**

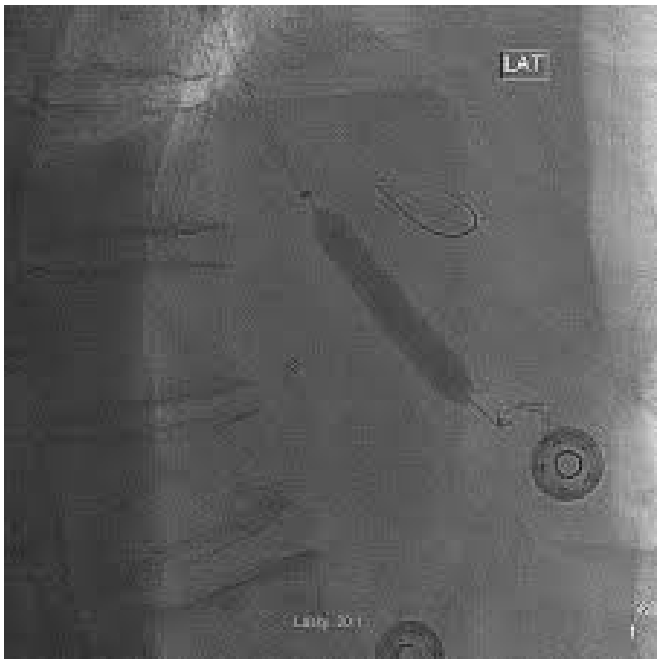



**Figure 13 Superior Mesenteric artery angioplasty**


**Figure 14 Post Procedure**

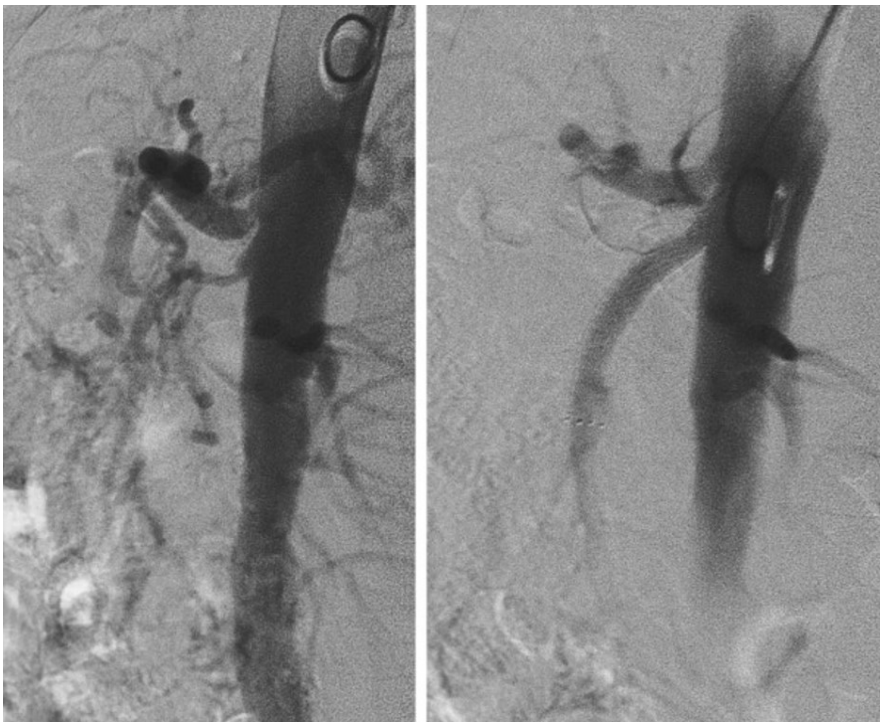


**Figure 15 Pre and Post Procedure angioplasty**

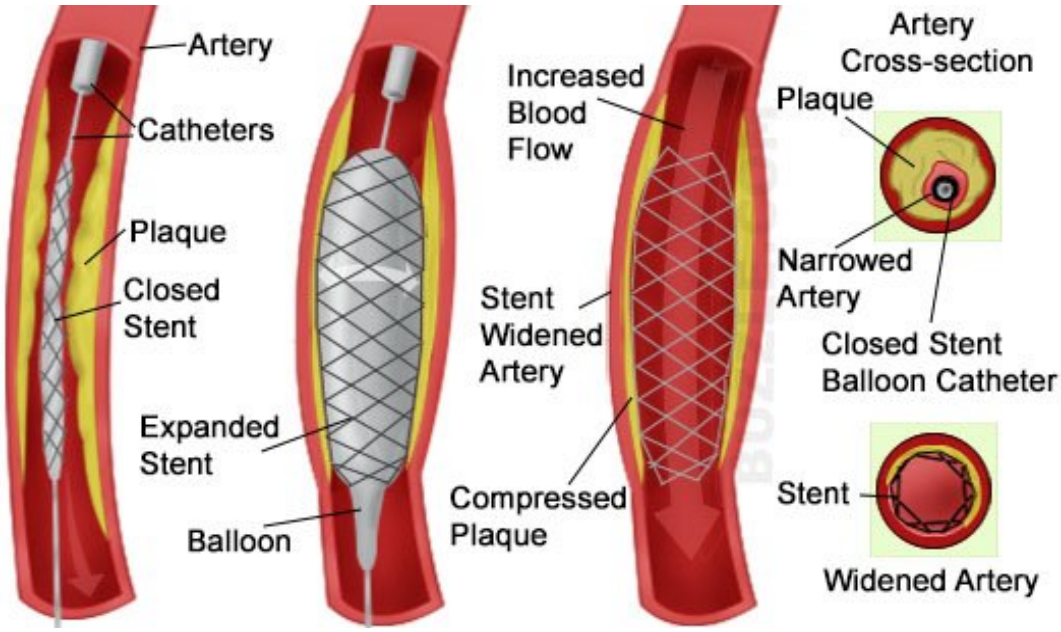



**Figure 16 Mechanism of Angioplasty and Stenting**

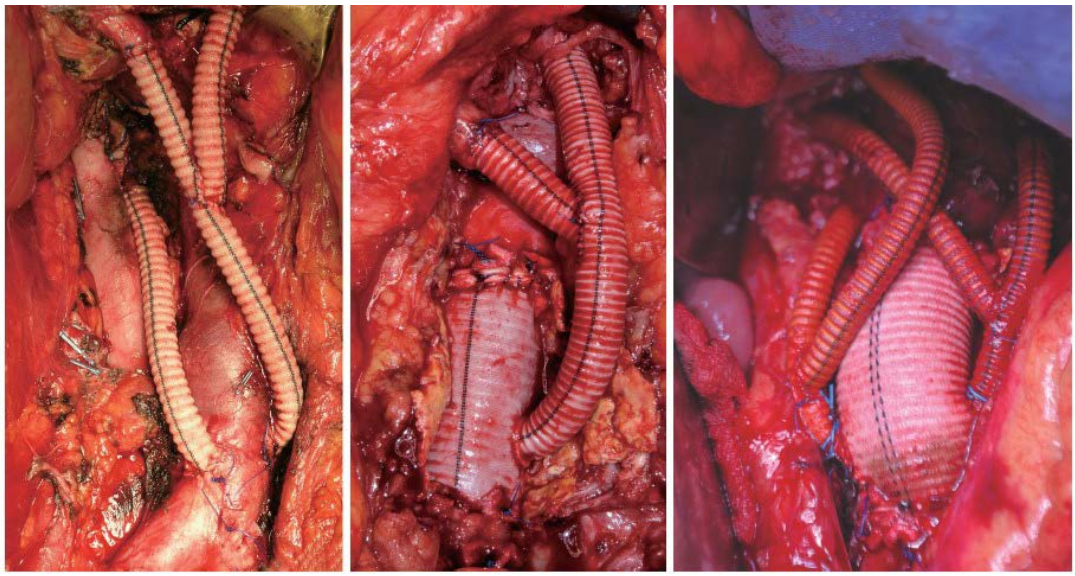


**Figure 17 Various procedures for open revascularization**

**Figure 18 Patient I - Critical Celiac axis stenosis with post-stenotic dilation**

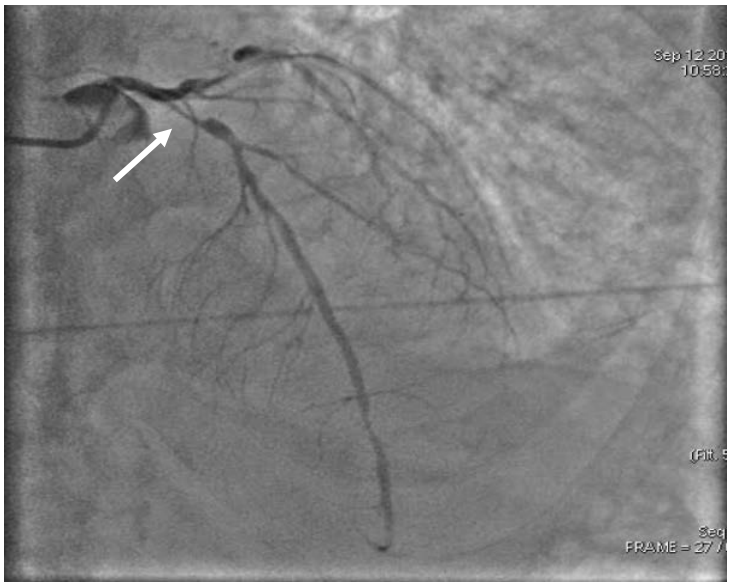


**Figure 19 Patient I - LAD ostio-proximal critical lesion with distal LAD involvement**

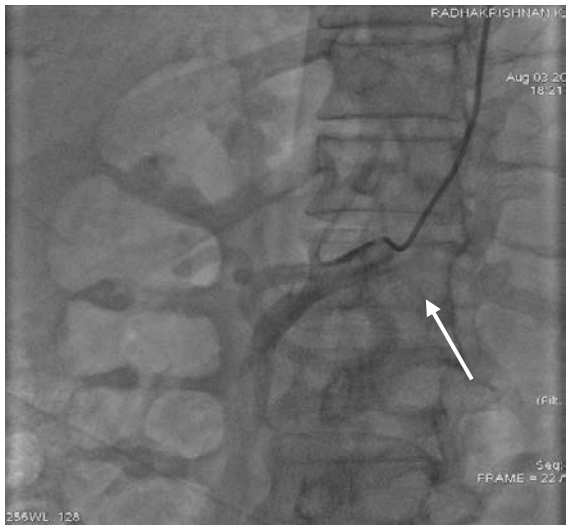


**Figure 20 Patient II - Common Origin of Celiac axis and Superior Mesenteric Artery - No stenosis**

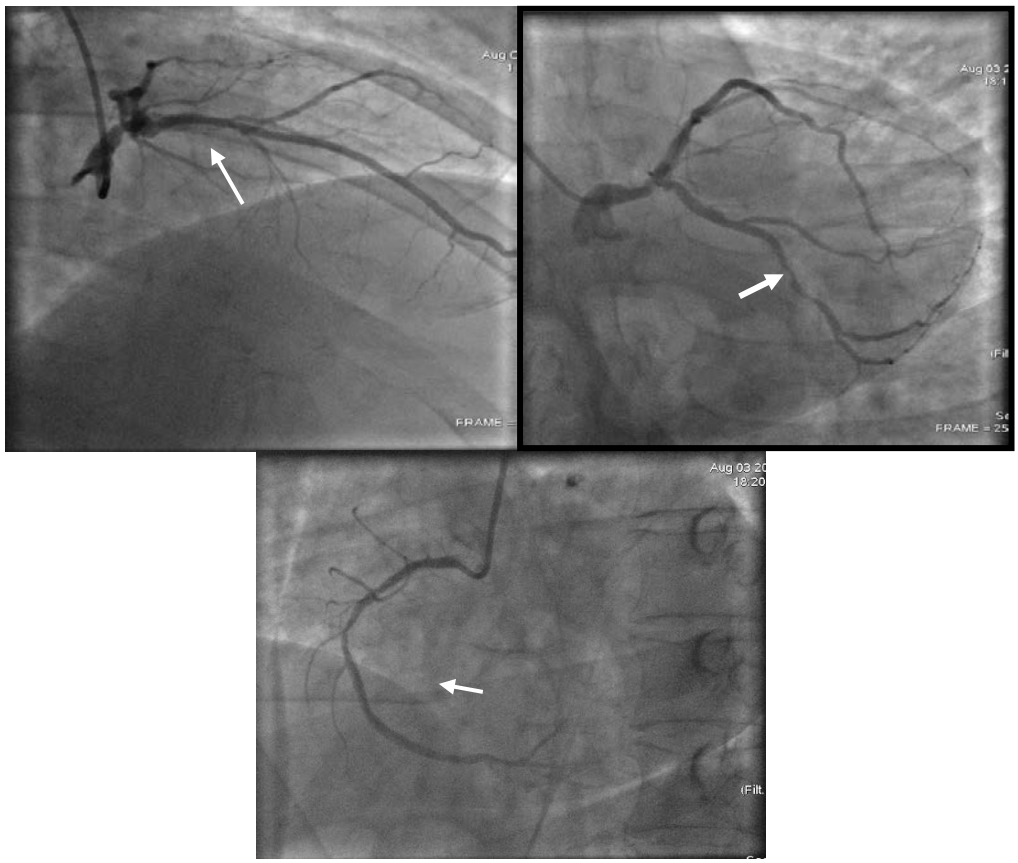


**Figure 21 - Patient II - Distal left main - 80% stenosis, Proximal Left Circumflex Artery - 90% stenosis, Proximal Right Circumflex Artery - 70% stenosis**

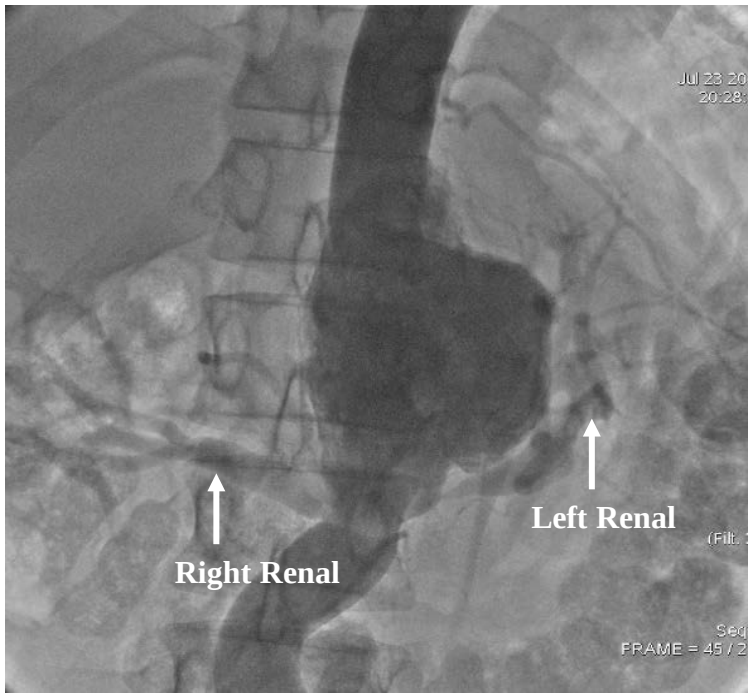



**Figure 22 - Patient III - AAA with Complete occlusion of Celiac axis and superior mesenteric artery origin - AP view**

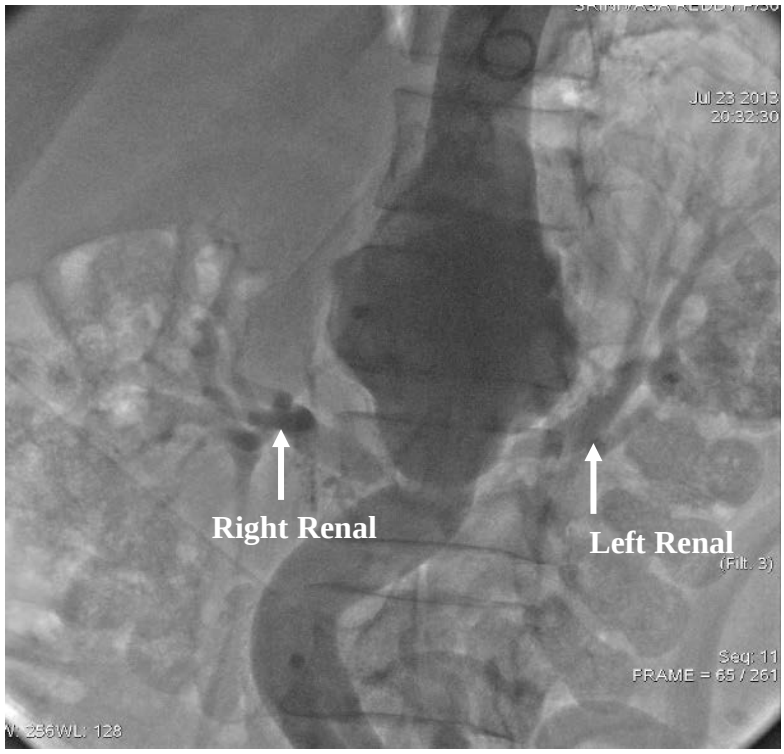



**Figure 23 - Patient III -AAA with Complete occlusion of Celiac axis and superior mesenteric artery origin - Lateral View**

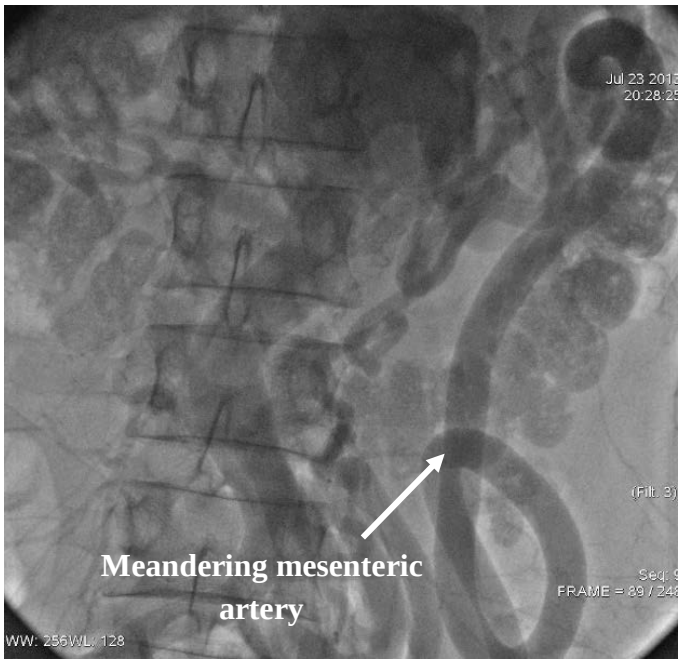



**Figure 24 - Patient III - Dilated Collateral arising from Inferior Mesenteric Artery**

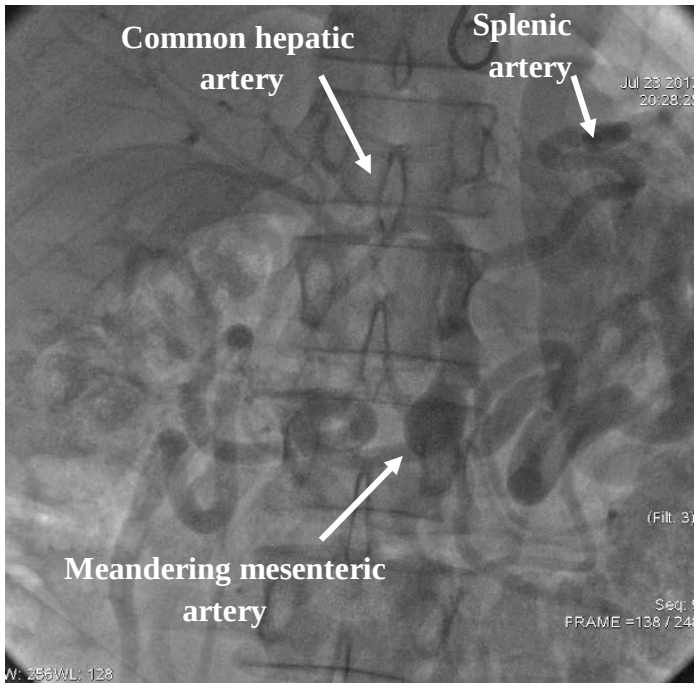



**Figure 25 - Patient III - Retrograde filling of Celiac Axis and Superior Mesenteric Artery**

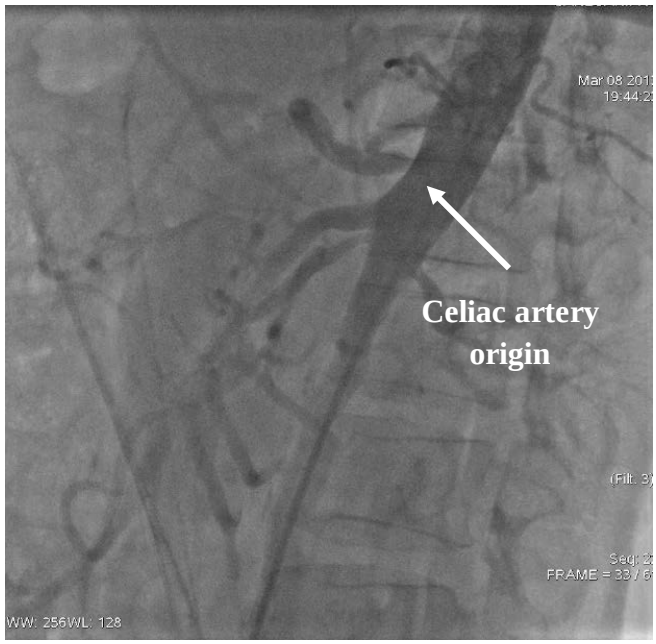



**Figure 26 - Patient IV - 90% Ostial stenosis of Celiac Axis and 30% stenosis of Proximal Superior Mesenteric Artery**

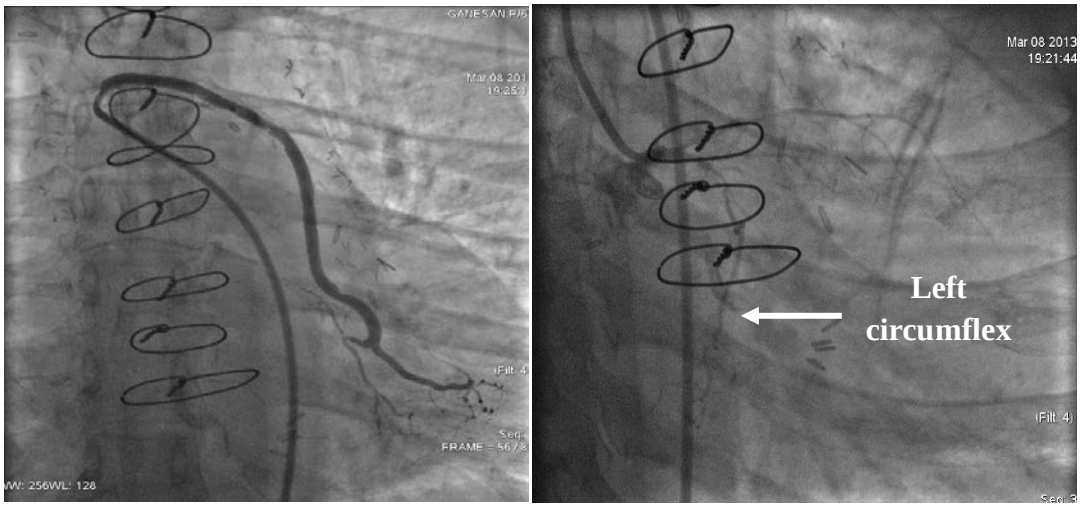



**Figure 27 - Patient IV - Post CABG - Patent Arterial Graft (Left internal thoracic artery to LAD) , Diffuse disease of LCX**

# RESULTS

From January 2013 to June 2013, a total of 110 patients underwent coronary angiogram for suspected ischemic heart disease in the Department of Cardiology at Govt. Stanley Medical College, Chennai. 7 patients were found to have normal coronary study and were excluded from the study. 103 patients were included for further analysis. Mesenteric angiogram revealed 42.7% (n=44) to have mesenteric vascular disease and 57.3% (n=59) with no mesenteric involvement.

The mean age of the population under study was 56.31 years with patients with and without mesenteric vessel involvement being 60.63 years and 53.08 years respectively. 64% (n=66) were males and 36% (n=37) were females. Only 26 (25%) patients had abdominal symptoms with weight loss being the major symptom. 7 out of these 26 had no evidence of mesenteric vascular disease. The prevalence of diabetes mellitus, hypertension, known ischemic heart disease and peripheral arterial disease among the study population was 40%, 22%, 22% and 20% respectively. Among those with mesenteric arterial involvement 70% were diabetic and 43% had prior history of peripheral arterial disease.

With respect to the coronary arteries the prevalence of single vessel, double vessel and triple disease was 36%, 43% and 21% respectively. Left Anterior descending artery was the most common involved coronary artery and affected in 73% (n=76) of the study population. Isolated left anterior descending artery affectation was present in 19 patients with isolated left circumflex and right coronary artery involvement in 14 and 5 patients respectively.

The prevalence of single vessel mesenteric disease and multivessel disease among the study population was 19.4% and 23.3% respectively.

Stenosis of superior mesenteric artery was present in 89% (n=39) of those with mesenteric involvement and 38% of the study population. Of these 39, isolated superior mesenteric artery stenosis was present in 16 patients and part of multi-vessel disease in 23 patients. The next common artery to be involved was celiac axis followed by inferior mesenteric artery in 22 and 15 patients respectively.

There was a strong association between presence of symptoms and superior mesenteric artery involvement.

Among the epidemiological factors age (65 years) and presence of abdominal symptoms were associated with prevalence of mesenteric vessel disease ($p<0.05$). Patients belonging to the underweight and

normal category was positively associated with mesenteric disease. With respect to the investigation, only serum albumin and erythrocyte sedimentation rate correlated with involvement of the mesenteric vessels.

The severity of the coronary disease was not associated with mesenteric vessel disease. However involvement of Left anterior descending artery indicated mesenteric vascular disease ($p=0.04$).

Among the 44 with mesenteric vascular disease, 18 fulfilled the criteria for revascularization of which 12 for open surgical revascularization ($p<0.01$). There was no significant association with a particular coronary artery with the need for revascularization.

# DISCUSSION

Atherosclerosis is a generalized phenomenon affecting all the vessels of the body especially the large and medium sized arteries. From being considered primarily a defect in fat metabolism, the theory behind its occurrence has evolved into an inflammatory process (57). Chronic inflammation is considered an essential component in the evolution of the atherosclerotic plaque starting from initiation to the eventual rupture of plaque (58). There have been reports of link between possible pathogens and the occurrence of atherosclerosis. The commonly implicated infectious agents include Chlamydia pneumonia and cytomegalovirus and their association is reinforced by their detection in human atherosclerotic lesion (59). The theory of atherosclerosis as being thought of inflammatory process has profound impact on the management of the disease. Though cannot be considered as substitute to traditional risk assessment, inflammatory markers, may prove to be useful adjuncts to determine the intensity of intervention (57). By the same concept, putative anti-inflammatory inhibitors like statins, ACE inhibitors, aspirin, and omega-3 fatty acids could have increased role.

Clinical studies have advocated that a state of “panvascular” active inflammation exists (60). In fact Lombardo et al in 2004 found that the not only was there a presence of concurrent atherosclerotic lesion in carotid artery in unstable angina patients but, the nature of coronary plaque instability correlated with carotid artery plaque instability. These observations have significant implications for understanding the mechanisms of acute widespread atherothrombotic plaque inflammation (61).

**Age**

Age plays a pivotal role in the process of atherosclerosis. It is associated with significant change in the physiological properties of the afflicted vessel (62). Increasing age is accompanied by marked intimal thickening and medial thinning of the peri-renal aorta (63). The age-related increase in intimal thickness is more pronounced in the abdominal aorta compared with other sites (64). Study by Jarvinen et al found that the prevalence of arterial abdominal stenosis in subjects younger than 40 years was 6%, in contrast to 67% in subjects aged above 80 years (65). Screening of asymptomatic elderly Americans (mean age 77 years) by duplex ultrasound demonstrated 18% involvement of the celiac axis or the superior mesenteric artery (66).

Roobottom et al found that in asymptomatic individuals the prevalence of isolated CA stenosis was 3% of subjects under 65 years and 18% in subjects above 65 years of age (67). This was concordant with our study with the mean of age of subjects with and without mesenteric involvement being 60.63 years and 53.08 years respectively. There was a significant difference between age groups <65 years and ≥ 65 years ($p<0.01$).

**Gender**

The involvement of aortic atherosclerosis in pathological specimen suggests that the advancement of atherosclerotic process occurs at a younger age in women concurring with clinical observation of peripheral arterial disease occurs earlier in females (63, 68). The preponderance to male in atherosclerotic disease is confined to the coronary vessels (45, 69). Veenstra et al in a study of 376 patients with chronic splanchnic ischemia found 74% of the affected population to be females (70). In our study there was no significant difference between the two genders with respect to mesenteric vascular disease or symptomatic mesenteric disease ($p=0.08$ & $p=0.8$). The difference is possibly due to the fact that the study population included patients who underwent coronary artery evaluation which is more common in males.

## Mesenteric artery disease

In 1981, Croft et al found in 203 autopsies that mean stenosis of celiac axis, superior mesenteric artery and the inferior mesenteric artery to be 21.9%, 27.3% and 30.5% respectively (71). In another series examining the asymptomatic elderly also showed out of 553 elderly, 97 had critical mesenteric arterial occlusion with isolated celiac axis and superior mesenteric involved in 83% and 5% respectively (55). In 45 patients who underwent stenting for symptomatic mesenteric arterial occlusion, 19 patients for superior mesenteric artery stenting, 8 for celiac axis stenting and 10 for both the vessels (72). The above studies probably indicate that there is high prevalence of celiac axis stenosis in the asymptomatic individuals whereas in symptomatic patients the superior mesenteric artery is the most common artery involved. The presence of abdominal symptoms suggestive of mesenteric insufficiency was significantly associated with presence of mesenteric vascular disease ($p<0.01$). Ischemic heart disease patients with abdominal symptoms were at 5 times more risk of having mesenteric vessel disease when compared to those without abdominal symptoms (OR = 5.64 95% CI: 2.06-15.09).

**Symptomatology**

The classical triad of symptoms of post prandial pain, weight loss and fear of food is seldom found. The fact that ischemic heart disease patients are on polypharmacy and have multiple co-morbidities makes it difficult for the treating physician and the patient to attribute vague abdominal symptomsat initial stages to mesenteric vascular insufficiency. As discussed earlier, involvement of the superior mesenteric artery is proposed to be primarily responsible for the symptomatology given its high prevalence in symptomatic patients on the contrary to the involvement of celiac axis in asymptomatic individuals. Wilson et al found that clinically significant loss of weight had a strong correlation with superior mesenteric artery stenosis (55). Weight loss was the predominant symptoms occurring in all the 26 symptomatic patients and classical triad present only in 4 patients. Of the 26 patients who had symptoms suggestive mesenteric insufficiency 16 of them had involvement of superior mesenteric artery ($p<0.01$). Other abdominal symptoms such as vomiting and diarrhea were rarely present.

## Co-morbid Diseases

*Diabetes mellitus*

One of the most important predisposing factor of atherosclerosis and cardiovascular disease, by corollary, plays a vital role in chronic mesenteric ischemia (31). The added insult to the intestinal blood supply by diabetic autonomic neuropathy exaggerates the gastrointestinal tract's inability to increase blood supply during the post prandial period. Veenstra et al showed 21% of chronic gastrointestinal ischemia patients had diabetes mellitus. Our study not only found that diabetic patients in the background of coronary artery disease were at 7 times more risk of developing mesenteric arterial disease (95% CI : 3.11-18.42) but also had 2.5 times the risk of needing vascular intervention to treat the mesenteric disease (95% CI : 0.89-6.82).

*Hypertension and Dyslipidemia*

Classical risk factors for cardiovascular disease, hypertension and dyslipidemia, have been reported by various studies to be associated with chronic gastrointestinal ischemia (49). Sara et al found that atherosclerotic chronic gastrointestinal ischemia were more likely to be suffering from hypercholesterolemia and hypertension when compared

to the control group (31, 49). In a study based in Netherlands the prevalence of hypercholesterolemia and hypertension was 53% and 62% respectively (70). In our study the prevalence of hypertension and dyslipidemia in patients with mesenteric vascular disease was 22% and 34% respectively ($p>0.05$).

*Peripheral arterial disease*

Being a process affecting large and medium sized arteries, vascular beds throughout the body are prone for affectation. This has been ascertained by various studies (70, 73, 74). Their strong association necessitates the screening for involvement of one vascular bed when the patient presents with atherosclerotic vascular insufficiency at one site. Carotid artery stenosis has been reported to range from 2-18% in asymptomatic screened population. However they can be as high 30% in high-risk population such as those with coronary artery disease (73). A study of 1405 patients with coronary artery disease revealed 5% prevalence of concomitant severe carotid artery disease (> 70% stenosis). A similar involvement of the renal artery (>75% stenosis) was also found in 3987 patients who underwent abdominal angiogram during coronary artery evaluation (74). On the same note, 9% of coronary artery disease patients had lower limb peripheral arterial disease and there was a

significant correlation to the extent of coronary artery disease (75). The reverse also holds good in that 59% of chronic mesenteric ischemia had atherosclerotic disease in other vascular beds (70). The past history of peripheral arterial disease was highly significant in our study with mesenteric vascular disease with a positive history predisposing the patient to 21 times the risk of having mesenteric vascular disease compared to negative history (95% CI : 4.68 - 100.13).

**Serum markers**

Identification of reliable serum markers for intestinal ischemia for chronic mesenteric ischemia would be of immense value as a non-invasive diagnostic test since the only test to test mucosal ischemia, tonometry, is limited by its availability and patient-unfriendly nature (35). Various serum markers have been described for acute mesenteric ischemia including early markers like intestinal fatty acid binding protein, D-dimer and late markers like lactate dehydrogenase, C-reactive protein (76). Research has been directed identifying these biomarkers by inducing ischemia, such as post prandial period, to identify chronic gastrointestinal insufficiency. In our study we found serum albumin to be significantly associated with mesenteric vessel disease probably due to malabsorption rather than a marker of intestinal ischemia.

## Mesenteric arterial disease

On analyzing the 103 patients, 44 patients had involvement of mesenteric arterial disease among which 19 had abdominal symptoms. Superior mesenteric artery was the most common artery involved either in isolation or as combination with other vessel involvement. There is lack of consensus as to vessel commonly involved, with studies in asymptomatic individuals pointing towards celiac axis and those studies based on population subjected to intervention tending towards superior mesenteric artery involvement (55, 66, 77).

Foley even suggested that revascularization of the superior mesenteric artery alone should suffice given its prominent role in symptomatology (78). This could be applied to our study population and gains clinical importance. Endovascular intervention for single mesenteric vessel, superior mesenteric artery, even in the presence of other mesenteric vessel involvement is feasible when angioplasty is planned for the coronary artery disease and can have good clinical outcome.

## Coronary Artery Disease

There have been studies intending to find correlation of the coronary artery disease and arterial diseases at other locations. The largest of

these is by Imori et al involving 1734 patients. The study was aimed at finding co-existence of coronary artery disease with other atherosclerotic vascular lesions involving the carotid, renal and lower limb peripheral artery. The studied demonstrated that majority of patients with coronary artery disease and association of other vascular beds, especially multivessel coronary artery disease. They suggested proactive screening for synchronous atherosclerotic vascular lesion at other sites in multivessel disease and involvement of left main disease (46). The reason for predisposition for left main disease to have concurrent lesion at other vascular beds has been poorly understood.

The left main coronary involvement is generally associated severe coronary artery disease in that 80% of these patients have multivessel coronary artery disease and 50% have right coronary involvement. Furthermore, at least total occlusion of one coronary artery was present in 50% of patients. All these factors make the left main involvement associated with unfavourable outcome (79).

In our study the involvement of left anterior descending artery was associated with mesenteric vessel disease ($p=0.04$). The severity of the coronary disease, single vessel disease or multi vessel disease was not associated with mesenteric vessel involvement ($p=0.14$).

# CONCLUSION

The process of atherosclerosis is a generalized process sparing no vascular bed. However the variability of sequence and severity of affectation at various sites poses challenge with respect to screening of distant vascular beds at the time of diagnosis of atherosclerotic vascular disease at one site.

Coronary artery disease is a pandemic disease and probably one of the first site to be involved in the process of atherosclerosis. Given the widespread awareness among the patients, well-formed guidelines for itsdiagnosis and treatment of coronary artery disease among the physician and the dramatic symptomatology, trend is towards detection at early stages and resulting in increased salvage of myocardium.

On the other hand atherosclerotic process of other vascular beds such as carotid artery and renal artery can be screened by simple, non-invasive, reliable duplex scan in patients with high risk factors.

Chronic mesenteric ischemia, if at all diagnosed, is identified at a late stage of the disease process. Lack of a simple reliable non-invasive tests in comparison to the gold standard invasive arteriogram prohibits the screening of high risk individuals. With recent studies highlighting the

reliability of non-invasive tests, single or in combination, would probably make the detection of mesenteric insufficiency at an earlier stage. Earlier there were reports that mesenteric arteries screening was unnecessary in asymptomatic individuals and results in unnecessary expenditure (55). However recent studies with better understanding of the natural course of the disease have been categorical with respect to need for intervention in asymptomatic individuals with significant mesenteric artery narrowing (53).

Our study clearly demonstrates the presence of abdominal symptoms in coronary artery disease patients is strongly associated with mesenteric disease. Advanced age, presence of diabetes mellitus and peripheral arterial disease are useful predictors of mesenteric vascular disease. Though the severity of the coronary artery disease is not associated with occurrence of mesenteric disease, the involvement of left anterior descending artery should necessitate the physician to rule out mesenteric vessel involvement.

Advances in the endovascular techniques, expertise and stents have seen the gap between a morbid open revascularization and relatively safe endovascular revascularization being narrowed with respect to their indication. Though the long term outcome of severe multivessel disease

is better with surgical revascularization, endovascular intervention has an important role in single vessel disease (51). The fact that superior mesenteric artery is considered as the primary culprit for the symptoms and revascularization of superior mesenteric artery alone may be adequate in treating chronic mesenteric insufficiency makes endovascular approach more attractive (78). Given the high prevalence of mesenteric vascular disease with or without symptoms in the background of atherosclerotic vascular disease at other sites, screening of the mesenteric vessel for involvement and also treating the same with endovascular intervention might increase the overall survival by reduction of acute mesenteric thrombosis.

# LIMITATIONS

Mucosal ischemia not confirmed by tonometry

Follow-up period too short to assess the natural course of the disease

# BIBLIOGRAPHY


1. Acosta S. Epidemiology of mesenteric vascular disease: clinical implications. Seminars in vascular surgery. 2010;23(1):4-8.
2. Anson BJ MC. Surgical Anatomy. 5 ed. Philadelphia: W.B. Saunders Co.; 1971.
3. Meschan I. Anatomy Basic to Radiology. . Philadelphia: W.B. Saunders Co.; 1975.
4. PL Williams RW, M Dyson, LH Bannister. Gray's Anatomy. Edinburgh: Churchill Livingstone; 1989.
5. Kornafel O, Baran B, Pawlikowska I, Laszczynski P, Guzinski M, Sasiadek M. Analysis of anatomical variations of the main arteries branching from the abdominal aorta, with 64-detector computed tomography. Polish Journal of Radiology. 2010;75(2):38-45.
6. Hines JR, Gore RM, Ballantyne GH. Superior mesenteric artery syndrome. Diagnostic criteria and therapeutic approaches. American journal of surgery. 1984;148(5):630-2.
7. Douard R, Chevallier JM, Delmas V, Cugnenc PH. Clinical interest of digestive arterial trunk anastomoses. Surgical and Radiologic Anatomy. 2006;28(3):219-27.
8. Gourley EJ, Gering SA. The meandering mesenteric artery: a historic review and surgical implications. Diseases of the colon and rectum. 2005;48(5):996-1000.
9. Ferro C, Rossi UG, Seitun S, Bovio G, Fornaro R. Endovascular treatment of totally occluded superior mesenteric artery by retrograde crossing via the Villemin arcade. Cardiovascular and interventional radiology. 2013;36(3):848-52.
10. AP Shepard DG. Physiology of the Intestinal Circulation. 6 ed. New York: Raven Press; 1984.
11. Moneta GL, Taylor DC, Helton WS, Mulholland MW, Strandness DE, Jr. Duplex ultrasound measurement of postprandial intestinal blood flow: effect of meal composition. Gastroenterology. 1988;95(5):1294-301.
12. Siregar H, Chou CC. Relative contribution of fat, protein, carbohydrate, and ethanol to intestinal hyperemia. The American journal of physiology. 1982;242(1):G27-31.
13. Bond JH, Prentiss RA, Levitt MD. The effects of feeding on blood flow to the stomach, small bowel, and colon of the conscious dog. The Journal of laboratory and clinical medicine. 1979;93(4):594-9.

14. Li KC, Whitney WS, McDonnell CH, Fredrickson JO, Pelc NJ, Dalman RL, et al. Chronic mesenteric ischemia: evaluation with phase-contrast cine MR imaging. Radiology. 1994;190(1):175-9.

15. Huber T S LWA, Seeger J M. Chronic mesenteric ischemia. In: Rutherford RB, editor. Vascular Surgery. 6 ed. Philadelphia: WB Saunders. p. 1732–47.

16. Poole JW, Sammartano RJ, Boley SJ. Hemodynamic basis of the pain of chronic mesenteric ischemia. American journal of surgery. 1987;153(2): 171-6.

17. Kruger AJ, Walker PJ, Foster WJ, Jenkins JS, Boyne NS, Jenkins J. Open surgery for atherosclerotic chronic mesenteric ischemia. Journal of vascular surgery : official publication, the Society for Vascular Surgery [and] International Society for Cardiovascular Surgery, North American Chapter. 2007;46(5):941-5.

18. Vogel TR. RUtherfordΓÇÖs vascular surgery. JAMA. 2011;306(20):2270-1.

19. Bradbury AW, Brittenden J, McBride K, Ruckley CV. Mesenteric ischaemia: a multidisciplinary approach. The British journal of surgery. 1995;82(11):1446-59.

20. Mansour MA. Management of acute mesenteric ischemia. Archives of surgery (Chicago, Ill : 1960). 1999;134(3):328-30; discussion 31.

21. Schoots IG, Koffeman GI, Legemate DA, Levi M, van Gulik TM. Systematic review of survival after acute mesenteric ischaemia according to disease aetiology. The British journal of surgery. 2004;91(1):17-27.

22. Weaver FA, Pentecost MJ, Yellin AE, Davis S, Finck E, Teitelbaum G. Clinical applications of carbon dioxide/digital subtraction arteriography. Journal of vascular surgery : official publication, the Society for Vascular Surgery [and] International Society for Cardiovascular Surgery, North American Chapter. 1991;13(2):266-72; discussion 72-3.

23. Seeger JM, Self S, Harward TR, Flynn TC, Hawkins IF. Carbon dioxide gas as an arterial contrast agent. Annals of Surgery. 1993;217(6):688-98.

24. Smith DC, Yahiku PY, Maloney MD, Hart KL. Three new low-osmolality contrast agents: a comparative study of patient discomfort. AJNR American journal of neuroradiology. 1988;9(1):137-9.

25. Lasser EC, Berry CC, Mishkin MM, Williamson B, Zheutlin N, Silverman JM. Pretreatment with corticosteroids to prevent adverse reactions to nonionic contrast media. AJR American journal of roentgenology. 1994;162(3):523-6.

26. Waybill MM, Waybill PN. Contrast media-induced nephrotoxicity: identification of patients at risk and algorithms for prevention. Journal of vascular and interventional radiology : JVIR. 2001;12(1):3-9.

27. Parfrey PS, Griffiths SM, Barrett BJ, Paul MD, Genge M, Withers J, et al. Contrast material-induced renal failure in patients with diabetes mellitus, renal insufficiency, or both. A prospective controlled study. The New England journal of medicine. 1989;320(3):143-9.

28. Rich MW, Crecelius CA. Incidence, risk factors, and clinical course of acute renal insufficiency after cardiac catheterization in patients 70 years of age or older. A prospective study. Archives of internal medicine. 1990;150(6):1237-42.

29. Gomes AS, Lois JF, Baker JD, McGlade CT, Bunnell DH, Hartzman S. Acute renal dysfunction in high-risk patients after angiography: comparison of ionic and nonionic contrast media. Radiology. 1989;170(1 Pt 1):65-8.

30. Lim HK, Lee WJ, Kim SH, Lee SJ, Choi SH, Park HS, et al. Splanchnic arterial stenosis or occlusion: diagnosis at Doppler US. Radiology. 1999;211(2):405-10.

31. Mensink PB, van Petersen AS, Geelkerken RH, Otte JA, Huisman AB, Kolkman JJ. Clinical significance of splanchnic artery stenosis. The British journal of surgery. 2006;93(11):1377-82.

32. Mensink PB, Geelkerken RH, Huisman AB, Kuipers EJ, Kolkman JJ. Twenty-four hour tonometry in patients suspected of chronic gastrointestinal ischemia. Digestive diseases and sciences. 2008;53(1):133-9.

33. Otte JA, Geelkerken RH, Huisman AB, Kolkman JJ. What is the best diagnostic approach for chronic gastrointestinal ischemia? The American journal of gastroenterology. 2007;102(9):2005-10.

34. Friedland S, Benaron D, Coogan S, Sze DY, Soetikno R. Diagnosis of chronic mesenteric ischemia by visible light spectroscopy during endoscopy. Gastrointestinal endoscopy. 2007;65(2):294-300.

35. van Noord D, Mensink PB, de Knegt RJ, Ouwendijk M, Francke J, van Vuuren AJ, et al. Serum markers and intestinal mucosal injury in chronic gastrointestinal ischemia. Digestive diseases and sciences. 2011;56(2): 506-12.

36. Nabel EG, Braunwald E. A tale of coronary artery disease and myocardial infarction. The New England journal of medicine. 2012;366(1):54-63.

37. Heberden W. Some account of a disorder of the breast. Medical Transactions 1772;2:59-67.

38. Hektoen L. Embolism of the left coronary artery; sudden death. Med Newsl (Lond) 1892;61(210).

39. Herrick JB. Landmark article (JAMA 1912). Clinical features of sudden obstruction of the coronary arteries. By James B. Herrick. JAMA : the journal of the American Medical Association. 1983;250(13):1757-65.

40. Felmeden D, Singh SP, Lip GY. Anomalous coronary arteries of aortic origin. International journal of clinical practice. 2000;54(6):390-4.

41. El-Menyar AA, Al Suwaidi J, Holmes DR, Jr. Left main coronary artery stenosis: state-of-the-art. Current problems in cardiology. 2007;32(3): 103-93.

42. Marzilli M, Merz CN, Boden WE, Bonow RO, Capozza PG, Chilian WM, et al. Obstructive coronary atherosclerosis and ischemic heart disease: an elusive link! Journal of the American College of Cardiology. 2012;60(11):951-6.

43. Fihn SD, Gardin JM, Abrams J, Berra K, Blankenship JC, Dallas AP, et al. 2012 ACCF/AHA/ACP/AATS/PCNA/SCAI/STS Guideline for the diagnosis and management of patients with stable ischemic heart disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American College of Physicians, American Association for Thoracic Surgery, Preventive Cardiovascular Nurses Association, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. Journal of the American College of Cardiology. 2012;60(24):e44-e164.

44. Fihn SD, Gardin JM, Abrams J, Berra K, Blankenship JC, Dallas AP, et al. 2012 ACCF/AHA/ACP/AATS/PCNA/SCAI/STS guideline for the diagnosis and management of patients with stable ischemic heart disease: executive summary: a report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines, and the American College of Physicians, American Association for Thoracic Surgery, Preventive Cardiovascular Nurses Association, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. Circulation. 2012;126(25):3097-137.

45. Chambless LE, Folsom AR, Davis V, Sharrett R, Heiss G, Sorlie P, et al. Risk factors for progression of common carotid atherosclerosis: the Atherosclerosis Risk in Communities Study, 1987-1998. American journal of epidemiology. 2002;155(1):38-47.

46. Imori Y, Akasaka T, Ochiai T, Oyama K, Tobita K, Shishido K, et al. Co-Existence of Carotid Artery Disease, Renal Artery Stenosis, and Lower Extremity Peripheral Arterial Disease in Patients With Coronary Artery Disease. The American journal of cardiology. 2013.

47. Bhatt DL, Peterson ED, Harrington RA, Ou FS, Cannon CP, Gibson CM, et al. Prior polyvascular disease: risk factor for adverse ischaemic outcomes in acute coronary syndromes. European heart journal. 2009;30(10):1195-202.

48. Alberts MJ, Bhatt DL, Mas JL, Ohman EM, Hirsch AT, Rother J, et al. Three-year follow-up and event rates in the international REduction of

Atherothrombosis for Continued Health Registry. European heart journal. 2009;30(19):2318-26.

49. Aria Sana DvN, Stephanie Kooij, Kim van Dijk, Bert Bravenboer, Louis Lieverse, Eric Sijbrands, Janneke Langendonk, Peter Mensink. Chronic Gastrointestinal Ischemia Due to Atherosclerotic Narrowing is Related to Classical Risk Factors for Cardiovascular Disease. Gastroenterology. May 2011;140(5):699.

50. Loffroy R, Steinmetz E, Guiu B, Molin V, Kretz B, Gagnaire A, et al. Role for endovascular therapy in chronic mesenteric ischemia. Canadian journal of gastroenterology=Journal canadien de gastroenterologie. 2009;23(5): 365-73.

51. Pecoraro F, Rancic Z, Lachat M, Mayer D, Amann-Vesti B, Pfammatter T, et al. Chronic mesenteric ischemia: critical review and guidelines for management. Annals of Vascular Surgery. 2013;27(1):113-22.

52. Zeller T, Rastan A, Sixt S. Chronic atherosclerotic mesenteric ischemia (CMI). Vascular medicine (London, England). 2010;15(4):333-8.

53. Thomas JH, Blake K, Pierce GE, Hermreck AS, Seigel E. The clinical course of asymptomatic mesenteric arterial stenosis. Journal of vascular surgery : official publication, the Society for Vascular Surgery [and] International Society for Cardiovascular Surgery, North American Chapter. 1998;27(5):840-4.

54. Stoney RJ, Cunningham CG. Acute mesenteric ischemia. Surgery. 1993;114(3):489-90.

55. Wilson DB, Mostafavi K, Craven TE, Ayerdi J, Edwards MS, Hansen KJ. Clinical course of mesenteric artery stenosis in elderly americans. Archives of internal medicine. 2006;166(19):2095-100.

56. Levy PJ, Krausz MM, Manny J. Acute mesenteric ischemia: improved results--a retrospective analysis of ninety-two patients. Surgery. 1990;107(4):372-80.

57. Montero-Vega MT. The inflammatory process underlying atherosclerosis. Critical reviews in immunology. 2012;32(5):373-462.

58. Rodolfo Paoletti AMG, Jr, David P. Hajjar. Inflammation in Atherosclerosis and Implications for Therapy. Circulation. 2004;109(III):20-6.

59. Gattone M, Iacoviello L, Colombo M, Castelnuovo AD, Soffiantino F, Gramoni A, et al. Chlamydia pneumoniae and cytomegalovirus seropositivity, inflammatory markers, and the risk of myocardial infarction at a young age. American heart journal. 2001;142(4):633-40.

60. Rothwell PM, Villagra R, Gibson R, Donders RC, Warlow CP. Evidence of a chronic systemic cause of instability of atherosclerotic plaques. Lancet. 2000;355(9197):19-24.

61. Lombardo A, Biasucci LM, Lanza GA, Coli S, Silvestri P, Cianflone D, et al. Inflammation as a possible link between coronary and carotid plaque instability. Circulation. 2004;109(25):3158-63.

62. O'Rourke MF. Arterial aging: pathophysiological principles. Vascular medicine (London, England). 2007;12(4):329-41.

63. van Dijk RA, Virmani R, von der Thusen JH, Schaapherder AF, Lindeman JH. The natural history of aortic atherosclerosis: a systematic histopathological evaluation of the peri-renal region. Atherosclerosis. 2010;210(1):100-6.

64. Henry Z. Movat RHM, M. Daria Haust. The Diffuse Intimal Thickening of the Human Aorta with Aging. American Journal of Pathology. 1958;34(6):1023-31.

65. Jarvinen O, Laurikka J, Sisto T, Salenius JP, Tarkka MR. Atherosclerosis of the visceral arteries. VASA Zeitschrift fur Gefasskrankheiten. 1995;24(1): 9-14.

66. Hansen KJ, Wilson DB, Craven TE, Pearce JD, English WP, Edwards MS, et al. Mesenteric artery disease in the elderly. Journal of vascular surgery : official publication, the Society for Vascular Surgery [and] International Society for Cardiovascular Surgery, North American Chapter. 2004;40(1):45-52.

67. Roobottom CA, Dubbins PA. Significant disease of the celiac and superior mesenteric arteries in asymptomatic patients: predictive value of Doppler sonography. AJR American journal of roentgenology. 1993;161(5):985-8.

68. Diehm C, Schuster A, Allenberg JR, Darius H, Haberl R, Lange S, et al. High prevalence of peripheral arterial disease and co-morbidity in 6880 primary care patients: cross-sectional study. Atherosclerosis. 2004;172(1):95-105.

69. Bauer M, Mohlenkamp S, Lehmann N, Schmermund A, Roggenbuck U, Moebus S, et al. The effect of age and risk factors on coronary and carotid artery atherosclerotic burden in males-Results of the Heinz Nixdorf Recall Study. Atherosclerosis. 2009;205(2):595-602.

70. Veenstra RP, ter Steege RW, Geelkerken RH, Huisman AB, Kolkman JJ. The cardiovascular risk profile of atherosclerotic gastrointestinal ischemia is different from other vascular beds. The American journal of medicine. 2012;125(4):394-8.

71. Croft RJ, Menon GP, Marston A. Does 'intestinal angina' exist? A critical study of obstructed visceral arteries. The British journal of surgery. 1981;68(5):316-8.

72. Aschenbach R, Bergert H, Kerl M, Zangos S, Neumeister A, Schlosser A, et al. Stenting of stenotic mesenteric arteries for symptomatic chronic mesenteric ischemia. VASA Zeitschrift fur Gefasskrankheiten. 2012;41(6):425-31.

73. Steinvil A, Sadeh B, Arbel Y, Justo D, Belei A, Borenstein N, et al. Prevalence and predictors of concomitant carotid and coronary artery atherosclerotic disease. Journal of the American College of Cardiology. 2011;57(7):779-83.

74. Conlon PJ, Little MA, Pieper K, Mark DB. Severity of renal vascular disease predicts mortality in patients undergoing coronary angiography. Kidney international. 2001;60(4):1490-7.

75. Makowsky MJ, McAlister FA, Galbraith PD, Southern DA, Ghali WA, Knudtson ML, et al. Lower extremity peripheral arterial disease in individuals with coronary artery disease: prognostic importance, care gaps, and impact of therapy. American heart journal. 2008;155(2):348-55.

76. Block T, Nilsson TK, Bjorck M, Acosta S. Diagnostic accuracy of plasma biomarkers for intestinal ischaemia. Scandinavian journal of clinical and laboratory investigation. 2008;68(3):242-8.

77. Zerbib P, Lebuffe G, Sergent-Baudson G, Chamatan A, Massouille D, Lions C, et al. Endovascular versus open revascularization for chronic mesenteric ischemia: a comparative study. Langenbeck's archives of surgery / Deutsche Gesellschaft fur Chirurgie. 2008;393(6):865-70.

78. Foley MI, Moneta GL, Abou-Zamzam AM, Jr., Edwards JM, Taylor LM, Jr., Yeager RA, et al. Revascularization of the superior mesenteric artery alone for treatment of intestinal ischemia. Journal of vascular surgery : official publication, the Society for Vascular Surgery [and] International Society for Cardiovascular Surgery, North American Chapter. 2000;32(1):37-47.

79. Gehani AA, El-Menyar A, Elgendy I, Abuzaid A, Ahmed E, Haque S. Clinical presentation and cardiovascular risk profiles in patients with left main coronary artery disease in a middle eastern country. Angiology. 2013;64(3):195-9.

# INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Prevalence of mesenteric vascular disease in patients with Coronary artery disease

Principal Investigator : Dr.K.Gautham

Designation : PG in MS (General Surgery)

Department : Department of General Surgery
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 08.04.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

13/9/13

MEMBER SECRETARY,
IEC, SMC, CHENNAI

# PROFORMA

- Name : sl. No:
- Age /sex:
- Address with contact number:

- Ip no:
- Date of admission: Date of discharge:

**History of presenting illness:**

**Cardiac Symptoms**

| | **Chest pain** | **SOB** | **Palpitation** | **Syncope** |
|---|---|---|---|---|
| Onset | | | | |
| Duration | | | | |
| Progression | | | | |
| Persistent/intermittent | | | | |

• Abdominal symptoms

Weight loss yes/no Quantification

Post prandial pain: yes/no

Fear of food yes/no

Nausea yes/no Vomiting yes/no

Constipation yes/no Diarrhea yes/no

**Past history:**

DM yes/no duration HT yes/no duration

IHD yes/no duration PVD yes/no duration

Dyslipidemia yes/no duration

**Personal history:**

Smoking yes/no Alcoholism yes/no

Physical activity low/moderate/high

**Family history:** yes/no

**Treatment history:** antiplatelet/ hypolipidemic agents/ OHA/ insulin/antianginal

**Clinical examination:**

Anthropometry :

BMI - Under weight / Normal / Overweight / Obese

Waist/Hip ratio - Low / Normal / High / Very High

Heart Rate : Blood Pressure : Temperature :

**Investigations:**

| | | | |
|---|---|---|---|
| Hb | | RBS | |
| TC | | BUN | |
| DC | | S. creatinine | |
| ESR | | S. bilurubin | |
| Platelet | | AST/ALT | |
| Hba1c | | S. protein | |
| Cholesterol | | TGL | |
| HDL | | LDL | |

ECG Chest x-ray Abdomen x-ray

Echocardiogram – LV dysfunction – none / mild / moderate / severe

RWMA – present / absent

Angiogram

| | | | |
|---|---|---|---|
| **RCA** | | **SMA** | |
| **LAD** | | **Celiac** | |
| **LCX** | | **IMA** | |

**Follow up:**

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# MASTER CHART

| S No | Name | Age | Sex | Weight loss | Post prandial | Fear of food | Nausea | Vomiting | Constipation | Diarrhea | Chest pain | SOB | Palpitation | Syncope | DM | HT | PVD | IHD | Dyslipidemia | Family H/O | Smoking | Alcohol | BMI | W/H ratio | HDL | LDL | TGL | Hb | TC | ESR | Platelet | HbA1c | FBS | BUN | S. creatinine | S.bilurubin | SGOT | SGPT | S.albumin | RCA | LAD | LCX | Celtac | SMA | IMA |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Bhuvaneshwari | 64 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 42 | 145 | 154 | 10.6 | 6800 | 25 | 3.6 | 7 | 86 | 28 | 0.8 | 0.7 | 25 | 28 | 4.7 | 1 | 1 | 1 | 1 | 1 | 1 |
| 2 | Devarajan | 47 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 40 | 130 | 145 | 10.8 | 7200 | 5 | 3.8 | 7.4 | 99 | 25 | 0.7 | 0.6 | 32 | 30 | 4.3 | 1 | 3 | 1 | 1 | 2 | 2 |
| 3 | Fathima | 65 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 45 | 125 | 150 | 11 | 5600 | 5 | 2.8 | 6.8 | 102 | 30 | 0.8 | 0.6 | 32 | 36 | 4.2 | 3 | 3 | 3 | 1 | 1 | 1 |
| 4 | Kumuthavalli | 65 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 3 | 3 | 50 | 150 | 125 | 11.6 | 5800 | 5 | 3.4 | 7.1 | 97 | 25 | 0.6 | 0.6 | 30 | 34 | 4.7 | 1 | 3 | 1 | 1 | 1 | 1 |
| 5 | Mangalam | 70 | 2 | 1 | 1 | 2 | 1 | 2 | 2 | 1 | 1 | 1 | 2 | 2 | 1 | 1 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 30 | 155 | 145 | 8.6 | 8600 | 20 | 3.4 | 6.5 | 124 | 25 | 0.6 | 0.7 | 60 | 80 | 3.3 | 3 | 3 | 3 | 3 | 3 | 1 |
| 6 | Meenakshi | 55 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 45 | 154 | 169 | 10.3 | 5200 | 10 | 2.8 | 6.5 | 110 | 24 | 0.7 | 0.6 | 40 | 35 | 4.9 | 1 | 1 | 1 | 1 | 1 | 2 |
| 7 | Rajendran | 55 | 1 | 0 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 30 | 124 | 145 | 11.3 | 5600 | 12 | 2.9 | 6.5 | 120 | 22 | 0.6 | 0.7 | 34 | 32 | 3.8 | 1 | 3 | 3 | 1 | 3 | 1 |
| 8 | Shanmugam | 65 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 3 | 40 | 120 | 150 | 10.6 | 8400 | 10 | 3.6 | 7 | 96 | 21 | 0.6 | 0.7 | 35 | 31 | 4.3 | 3 | 3 | 1 | 1 | 1 | 1 |
| 9 | Beena | 45 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 3 | 50 | 130 | 150 | 10.4 | 4600 | 8 | 4 | 7.2 | 102 | 20 | 0.8 | 0.6 | 45 | 50 | 4 | 3 | 1 | 2 | 1 | 1 | 1 |
| 10 | Ramanaiah | 45 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 1 | 38 | 145 | 154 | 12.1 | 4900 | 5 | 3.7 | 7.1 | 100 | 24 | 0.5 | 0.5 | 24 | 26 | 3.9 | 3 | 2 | 1 | 1 | 1 | 1 |
| 11 | Sugurani | 58 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 35 | 125 | 120 | 11.8 | 6500 | 8 | 3.5 | 7.2 | 95 | 24 | 0.5 | 0.6 | 28 | 30 | 4.5 | 1 | 1 | 2 | 1 | 1 | 1 |
| 12 | Somarajan | 56 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 3 | 3 | 48 | 150 | 164 | 10.5 | 4700 | 8 | 3.5 | 6.5 | 97 | 22 | 0.5 | 0.9 | 24 | 24 | 4 | 3 | 3 | 1 | 1 | 1 | 1 |
| 13 | Sreekanth | 45 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 1 | 40 | 120 | 150 | 11.5 | 5900 | 6 | 3.8 | 8 | 102 | 25 | 0.6 | 0.6 | 28 | 29 | 3.5 | 2 | 3 | 1 | 2 | 2 | 2 |
| 14 | Swaminathan | 72 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 1 | 2 | 1 | 1 | 2 | 2 | 1 | 2 | 1 | 2 | 48 | 101 | 140 | 11 | 7800 | 12 | 2.5 | 6.9 | 123 | 28 | 0.8 | 0.6 | 24 | 28 | 3.3 | 3 | 3 | 3 | 2 | 2 | 1 |
| 15 | Theresa | 58 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 3 | 2 | 45 | 154 | 187 | 11.4 | 7400 | 10 | 3.4 | 6.5 | 103 | 24 | 0.5 | 0.6 | 28 | 30 | 3.1 | 3 | 2 | 1 | 1 | 1 | 1 |
| 16 | Vadugunathan | 49 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 2 | 3 | 2 | 30 | 145 | 168 | 11 | 6300 | 5 | 3.9 | 8.2 | 159 | 28 | 0.8 | 0.9 | 36 | 38 | 4 | 1 | 1 | 1 | 1 | 1 | 1 |
| 17 | Ganesan | 60 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 1 | 2 | 2 | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 3 | 2 | 50 | 145 | 126 | 11.4 | 9800 | 10 | 4.2 | 6.9 | 124 | 24 | 0.6 | 0.8 | 27 | 26 | 4.7 | 3 | 3 | 3 | 2 | 3 | 2 |
| 18 | Hemalatha | 58 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 3 | 2 | 45 | 136 | 182 | 10.8 | 7500 | 8 | 3.6 | 8.2 | 187 | 24 | 0.8 | 0.7 | 32 | 34 | 5.3 | 1 | 3 | 1 | 1 | 1 | 2 |
| 19 | Kanakaraj | 68 | 1 | 0 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 34 | 124 | 156 | 9.8 | 4500 | 12 | 4.5 | 7 | 101 | 24 | 0.5 | 0.6 | 25 | 28 | 3.1 | 1 | 2 | 1 | 1 | 1 | 1 |
| 20 | Leelavathy | 63 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 3 | 3 | 45 | 186 | 199 | 11.4 | 4900 | 5 | 3.8 | 7.8 | 145 | 28 | 0.7 | 0.6 | 21 | 29 | 5.3 | 1 | 1 | 1 | 1 | 1 | 1 |
| 21 | Madusudhana Rao | 65 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 2 | 2 | 1 | 2 | 1 | 2 | 2 | 1 | 1 | 3 | 3 | 42 | 120 | 140 | 11.4 | 8500 | 5 | 3.2 | 7.1 | 87 | 21 | 0.5 | 0.6 | 24 | 26 | 4.2 | 3 | 3 | 3 | 1 | 1 | 1 |
| 22 | Marichamy | 49 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 46 | 124 | 185 | 11.5 | 4500 | 5 | 3.6 | 6.8 | 89 | 24 | 0.8 | 0.6 | 24 | 28 | 3.6 | 3 | 3 | 3 | 1 | 1 | 1 |
| 23 | Mariyappan | 60 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 1 | 2 | 1 | 2 | 34 | 145 | 158 | 11.2 | 6500 | 7 | 3.8 | 6.8 | 86 | 35 | 0.7 | 0.8 | 25 | 30 | 4.7 | 1 | 1 | 2 | 1 | 2 | 1 |
| 24 | Premkumar | 48 | 1 | 0 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 3 | 3 | 45 | 142 | 157 | 11.6 | 8900 | 8 | 3.5 | 7.2 | 101 | 24 | 0.5 | 0.4 | 24 | 24 | 4.8 | 1 | 3 | 3 | 1 | 2 | 2 |
| 25 | Robert | 55 | 1 | 1 | 1 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 3 | 3 | 47 | 124 | 156 | 10.8 | 6500 | 7 | 4.2 | 6.3 | 104 | 25 | 0.6 | 0.6 | 80 | 120 | 4 | 3 | 3 | 3 | 1 | 2 | 1 |
| 26 | Siromani | 50 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 1 | 2 | 1 | 2 | 2 | 2 | 1 | 3 | 2 | 41 | 157 | 145 | 11.5 | 9840 | 5 | 3.6 | 7.1 | 88 | 26 | 0.8 | 0.6 | 24 | 36 | 4.3 | 3 | 2 | 1 | 1 | 1 | 1 |
| 27 | Subbaiah | 63 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 1 | 2 | 1 | 2 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 55 | 180 | 164 | 11.4 | 8500 | 12 | 3.5 | 7.2 | 87 | 25 | 0.5 | 0.8 | 84 | 96 | 2.9 | 3 | 3 | 3 | 3 | 3 | 2 |
| 28 | Velvizhi | 38 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 54 | 145 | 187 | 10.2 | 4850 | 10 | 3.5 | 7.2 | 98 | 24 | 0.8 | 0.7 | 87 | 102 | 3 | 1 | 1 | 3 | 2 | 3 | 2 |
| 29 | Vijayakumar | 44 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 3 | 47 | 142 | 168 | 11.4 | 8400 | 8 | 3.7 | 7 | 84 | 21 | 0.6 | 0.6 | 28 | 32 | 4.9 | 3 | 3 | 1 | 1 | 1 | 1 |
| 30 | Abdul Rahman | 59 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 1 | 2 | 3 | 3 | 45 | 124 | 169 | 11.1 | 5200 | 7 | 3.1 | 7.1 | 99 | 25 | 0.5 | 0.8 | 40 | 40 | 4.7 | 3 | 3 | 1 | 1 | 1 | 1 |
| 31 | Amsaveni Selvaraj | 63 | 1 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 40 | 145 | 165 | 8.6 | 9800 | 12 | 3.6 | 8.4 | 154 | 25 | 0.9 | 0.5 | 84 | 112 | 2.9 | 3 | 3 | 1 | 3 | 1 | 2 |
| 32 | Arumugam | 70 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 1 | 2 | 2 | 35 | 180 | 160 | 10.4 | 4560 | 10 | 3.6 | 7.2 | 102 | 25 | 0.4 | 0.5 | 24 | 26 | 3.3 | 1 | 3 | 3 | 2 | 2 | 2 |
| 33 | Ashok Kumar | 45 | 1 | 0 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 1 | 3 | 3 | 45 | 148 | 175 | 10.5 | 6800 | 12 | 3.4 | 7 | 104 | 24 | 0.5 | 0.6 | 65 | 84 | 4.7 | 1 | 2 | 3 | 1 | 1 | 1 |
| 34 | Bhaskaran | 60 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 3 | 3 | 40 | 152 | 160 | 10.5 | 7500 | 5 | 3 | 6.9 | 88 | 24 | 0.6 | 0.5 | 28 | 32 | 4.6 | 1 | 1 | 2 | 1 | 1 | 1 |
| 35 | Ganesan | 55 | 1 | 0 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 3 | 3 | 54 | 110 | 180 | 11.4 | 4900 | 10 | 2.8 | 7.2 | 98 | 28 | 0.5 | 0.6 | 44 | 52 | 5 | 1 | 2 | 2 | 1 | 1 | 1 |
| 36 | Girija | 69 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 51 | 175 | 142 | 10.5 | 7540 | 12 | 2.5 | 7 | 101 | 23 | 0.5 | 0.6 | 44 | 68 | 4.6 | 1 | 2 | 1 | 1 | 2 | 1 |
| 37 | Hulasi Bai | 70 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 47 | 157 | 168 | 8.5 | 6830 | 8 | 2.9 | 8.2 | 187 | 25 | 0.8 | 0.9 | 65 | 80 | 3.3 | 2 | 3 | 3 | 1 | 3 | 3 |
| 38 | Kuupu Rao | 65 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 3 | 3 | 30 | 195 | ## | 11.8 | 5830 | 8 | 3.6 | 7.2 | 124 | 26 | 0.6 | 0.9 | 42 | 45 | 3.7 | 3 | 3 | 3 | 1 | 3 | 1 |
| 39 | Mahaboor Basha | 37 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 40 | 120 | 157 | 10.2 | 7600 | 10 | 3.4 | 7.1 | 100 | 28 | 0.7 | 0.9 | 34 | 38 | 3.5 | 3 | 3 | 3 | 3 | 2 | 1 |
| 40 | Sabapathy | 64 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 1 | 2 | 2 | 45 | 150 | 180 | 10.5 | 8600 | 12 | 3.8 | 7.2 | 120 | 24 | 0.6 | 0.8 | 75 | 85 | 4 | 1 | 3 | 1 | 1 | 2 | 1 |

| S No | Name | Age | Sex | Weight loss | Post prandial | Fear of food | Nausea | Vomiting | Constipation | Diarrhea | Chest pain | SOB | Palpitation | Syncope | DM | HT | PVD | IHD | Dyslipidemia | Family H/O | Smoking | Alcohol | BMI | W/H ratio | HDL | LDL | TGL | Hb | TC | ESR | Platelet | HbA1c | FBS | BUN | S. creatinine | S.bilurubin | SGOT | SGPT | S.albumin | RCA | LAD | LCX | Celiac | SMA | IMA |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 41 | Samsudeen | 45 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 3 | 3 | 40 | 150 | 140 | 11.1 | 5900 | 6 | 3.4 | 6.8 | 85 | 36 | 0.4 | 0.9 | 40 | 85 | 5 | 1 | 3 | 1 | 1 | 1 | 1 |
| 42 | Saroja | 65 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 3 | 47 | 120 | ## | 9.8 | 6200 | 20 | 3.8 | 7.2 | 140 | 25 | 0.6 | 0.8 | 84 | 98 | 5 | 1 | 3 | 3 | 2 | 1 | 1 |
| 43 | Vijayabaskar | 70 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 3 | 3 | 45 | 15 | 124 | 11.2 | 3500 | 10 | 3.6 | 6.5 | 120 | 25 | 0.6 | 0.7 | 58 | 64 | 4.6 | 1 | 1 | 3 | 1 | 1 | 1 |
| 44 | Bala Thandayudham | 47 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 3 | 35 | 100 | 150 | 11.2 | 6800 | 12 | 2.8 | 6.5 | 96 | 28 | 0.8 | 0.8 | 28 | 30 | 4.7 | 1 | 1 | 1 | 1 | 1 | 1 |
| 45 | Dasari Devasree | 43 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 3 | 40 | 120 | 155 | 10.8 | 8600 | 5 | 2.9 | 7 | 85 | 20 | 0.9 | 0.9 | 30 | 35 | 4.6 | 2 | 2 | 1 | 1 | 1 | 1 |
| 46 | Gowri Shankar | 52 | 1 | 0 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 3 | 3 | 45 | 175 | 190 | 10.6 | 5800 | 6 | 2.6 | 7.6 | 138 | 22 | 0.9 | 0.9 | 30 | 35 | 4.3 | 3 | 1 | 3 | 1 | 1 | 1 |
| 47 | Durai Rajan | 77 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 1 | 2 | 45 | 135 | 150 | 9.8 | 6500 | 15 | 3.5 | 8 | 158 | 20 | 0.6 | 0.9 | 23 | 22 | 3.3 | 3 | 3 | 3 | 3 | 2 | 2 |
| 48 | Hemavathy | 59 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 50 | 130 | 150 | 10.9 | 7500 | 5 | 3.1 | 7 | 101 | 28 | 0.6 | 0.6 | 28 | 32 | 4.6 | 1 | 1 | 2 | 1 | 1 | 1 |
| 49 | Josephine | 62 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 40 | 120 | 115 | 11.6 | 8500 | 10 | 3.6 | 7.5 | 96 | 21 | 0.6 | 0.9 | 28 | 32 | 5.1 | 3 | 3 | 3 | 1 | 1 | 1 |
| 50 | Krishnamurthy | 69 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 2 | 2 | 3 | 40 | 140 | 150 | 11.1 | 5600 | 10 | 3.8 | 7 | 98 | 20 | 0.8 | 0.9 | 30 | 35 | 4 | 3 | 3 | 3 | 1 | 1 | 1 |
| 51 | Kundanrai Malhotra | 81 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 1 | 2 | 2 | 2 | 1 | 45 | 135 | 128 | 9.8 | 7500 | 22 | 3.6 | 7.1 | 86 | 26 | 0.8 | 0.6 | 24 | 32 | 3.1 | 2 | 2 | 3 | 3 | 3 | 2 |
| 52 | Meenakshiamma | 52 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 45 | 95 | 130 | 10.1 | 6800 | 12 | 2.8 | 72 | 98 | 22 | 0.6 | 0.9 | 25 | 30 | 5.4 | 1 | 1 | 2 | 1 | 1 | 1 |
| 53 | Murugan | 48 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 43 | 117 | 180 | 9.6 | 6800 | 15 | 2.6 | 7.1 | 85 | 29 | 0.6 | 0.9 | 30 | 35 | 4.7 | 3 | 3 | 3 | 1 | 1 | 1 |
| 54 | Nagaraja Rao | 67 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 1 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 3 | 3 | 42 | 158 | 140 | 10.8 | 8600 | 8 | 3.8 | 7 | 85 | 28 | 0.7 | 0.6 | 25 | 35 | 5.3 | 1 | 1 | 1 | 1 | 1 | 1 |
| 55 | Radhakrishnan | 56 | 1 | 1 | 1 | 2 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 45 | 145 | 180 | 10.6 | 6500 | 15 | 2.8 | 7.8 | 152 | 25 | 0.6 | 0.6 | 28 | 32 | 4 | 3 | 3 | 3 | 2 | 2 | 1 |
| 56 | Rajamani | 50 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 3 | 3 | 45 | 140 | 160 | 11.6 | 6800 | 10 | 3.8 | 7.6 | 85 | 25 | 0.6 | 0.8 | 20 | 25 | 5.1 | 3 | 3 | 1 | 1 | 1 | 1 |
| 57 | Ramakrishnaiah | 73 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 3 | 2 | 35 | 125 | 180 | 11.8 | 5800 | 5 | 1.8 | 6.5 | 80 | 24 | 0.6 | 0.6 | 35 | 30 | 3.3 | 3 | 3 | 3 | 1 | 1 | 1 |
| 58 | Somasundaram | 57 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 40 | 140 | 190 | 10.8 | 8600 | 5 | 3.6 | 8 | 85 | 28 | 0.6 | 0.6 | 30 | 35 | 3.5 | 3 | 1 | 1 | 1 | 3 | 1 |
| 59 | Srinvasa Reddy | 30 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 1 | 1 | 40 | 178 | ## | 10.8 | 8600 | 5 | 1.8 | 6.8 | 96 | 28 | 0.8 | 0.6 | 28 | 32 | 3.1 | 1 | 1 | 1 | 3 | 3 | 1 |
| 60 | Subramaniyam | 47 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 1 | 2 | 3 | 2 | 30 | 150 | 155 | 11 | 5800 | 5 | 3.6 | 6.8 | 101 | 28 | 0.7 | 0.7 | 35 | 32 | 4.4 | 3 | 1 | 3 | 1 | 1 | 1 |
| 61 | Yasodha | 62 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 40 | 120 | 180 | 9.8 | 8600 | 5 | 3.2 | 7.1 | 102 | 24 | 0.5 | 0.6 | 38 | 35 | 4.1 | 1 | 2 | 2 | 1 | 1 | 1 |
| 62 | Sabapathy | 45 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 2 | 45 | 124 | 159 | 11 | 6500 | 5 | 3.6 | 7 | 85 | 25 | 0.6 | 0.9 | 48 | 46 | 4.6 | 1 | 1 | 2 | 1 | 1 | 1 |
| 63 | Anajaneyalu | 48 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 1 | 3 | 2 | 40 | 135 | 140 | 11.2 | 4500 | 8 | 3.5 | 6.8 | 89 | 25 | 0.6 | 0.9 | 40 | 35 | 4 | 1 | 2 | 1 | 1 | 1 | 1 |
| 64 | Thangaraj | 65 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 1 | 1 | 2 | 1 | 42 | 112 | 130 | 11.4 | 8400 | 7 | 4.1 | 7.2 | 105 | 24 | 0.8 | 0.5 | 54 | 65 | 4.7 | 1 | 2 | 1 | 1 | 2 | 1 |
| 65 | Fathima Mary | 72 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 56 | 120 | 114 | 10.5 | 7500 | 22 | 3.4 | 7.9 | 120 | 21 | 0.5 | 0.6 | 25 | 40 | 4.3 | 1 | 2 | 3 | 2 | 3 | 1 |
| 66 | Mathiazhagan | 51 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 3 | 2 | 40 | 100 | 140 | 11.5 | 6500 | 8 | 3.6 | 6.5 | 112 | 25 | 0.6 | 0.8 | 42 | 40 | 4 | 1 | 2 | 1 | 1 | 1 | 1 |
| 67 | Sivashankar | 56 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 1 | 35 | 120 | 114 | 10.8 | 8500 | 4 | 3.5 | 6.5 | 98 | 25 | 0.6 | 0.9 | 58 | 68 | 4.2 | 1 | 3 | 2 | 1 | 3 | 1 |
| 68 | Tamil Selvi | 65 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 35 | 98 | 124 | 11.3 | 7500 | 8 | 3.1 | 7.4 | 98 | 24 | 0.5 | 0.6 | 40 | 43 | 3.6 | 1 | 3 | 2 | 3 | 1 |
| 69 | Mohan Krishnan | 35 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 3 | 48 | 112 | 145 | 11.8 | 7600 | 4 | 3.6 | 7.1 | 91 | 21 | 0.5 | 0.6 | 40 | 35 | 4.5 | 1 | 2 | 1 | 1 | 1 | 1 |
| 70 | Velayutham | 54 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 3 | 3 | 45 | 124 | 185 | 10.5 | 8400 | 8 | 3.9 | 7.5 | 109 | 24 | 0.5 | 0.7 | 35 | 39 | 4.6 | 1 | 1 | 2 | 1 | 1 | 1 |
| 71 | Mythiliraghavan | 40 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 3 | 40 | 120 | 142 | 10.8 | 8300 | 5 | 3.6 | 7 | 85 | 25 | 0.6 | 0.4 | 80 | 90 | 5 | 1 | 3 | 2 | 1 | 1 | 1 |
| 72 | Sudhakar Reddy | 62 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 1 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 42 | 115 | 124 | 11.5 | 4500 | 9 | 3.9 | 7.1 | 96 | 25 | 0.6 | 0.5 | 28 | 32 | 3.6 | 1 | 3 | 3 | 1 | 3 | 3 |
| 73 | Muniraghavan | 54 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 3 | 3 | 35 | 120 | 135 | 11.6 | 5400 | 5 | 3.1 | 6.8 | 94 | 24 | 0.9 | 0.8 | 23 | 28 | 3.9 | 2 | 2 | 2 | 1 | 1 | 1 |
| 74 | Thirugnanasambadam | 48 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 1 | 1 | 2 | 1 | 2 | 2 | 2 | 39 | 120 | 152 | 11.8 | 6500 | 8 | 3.8 | 6.5 | 94 | 28 | 0.9 | 0.6 | 40 | 35 | 4.6 | 1 | 2 | 2 | 1 | 1 | 1 |
| 75 | Sampathi | 42 | 2 | 0 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 3 | 2 | 36 | 114 | 125 | 10.8 | 7400 | 5 | 3.2 | 6.8 | 101 | 25 | 0.6 | 0.5 | 40 | 60 | 4.9 | 2 | 1 | 3 | 1 | 1 | 1 |
| 76 | Leelavathi Bai | 65 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 3 | 2 | 36 | 145 | 120 | 10.6 | 6500 | 12 | 3.4 | 6.5 | 104 | 25 | 0.8 | 0.4 | 24 | 28 | 3.3 | 2 | 3 | 1 | 2 | 3 | 1 |
| 77 | Thayumanavan | 45 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 35 | 124 | 165 | 11.5 | 5200 | 8 | 3.4 | 6.5 | 102 | 28 | 0.6 | 0.9 | 30 | 34 | 3.3 | 2 | 1 | 3 | 1 | 1 | 1 |
| 78 | Alamelu Mangai | 65 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 45 | 132 | 124 | 10.5 | 4500 | 10 | 3.5 | 6.8 | 98 | 24 | 0.6 | 0.5 | 45 | 42 | 3 | 2 | 2 | 2 | 2 | 3 | 2 |
| 79 | Jayamani | 55 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 3 | 42 | 110 | 110 | 11.2 | 4500 | 12 | 3.5 | 6.9 | 99 | 25 | 0.4 | 0.6 | 24 | 26 | 3.3 | 2 | 2 | 2 | 1 | 1 | 1 |
| 80 | Boopalan | 45 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 3 | 3 | 48 | 110 | 142 | 11.4 | 8500 | 6 | 3.5 | 7.6 | 140 | 25 | 0.5 | 0.6 | 40 | 35 | 4.3 | 1 | 2 | 1 | 1 | 1 | 1 |

| S No | Name | Age | Sex | Weight loss | Post prandial | Fear of food | Nausea | Vomiting | Constipation | Diarrhea | Chest pain | SOB | Palpitation | Syncope | DM | HT | PVD | IHD | Dyslipidemia | Family H/O | Smoking | Alcohol | BMI | W/H ratio | HDL | LDL | TGL | Hb | TC | ESR | Platelet | HbA1c | FBS | BUN | S. creatinine | S.bilurubin | SGOT | SGPT | S.albumin | RCA | LAD | LCX | Celiac | SMA | IMA |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 81 | Murali | 45 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 3 | 2 | 42 | 105 | 120 | 11.8 | 6500 | 5 | 3.2 | 6.8 | 96 | 24 | 0.6 | 0.9 | 65 | 85 | 4.6 | 1 | 2 | 1 | 1 | 1 | 1 |
| 82 | Prathap Reddy | 56 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 1 | 3 | 2 | 56 | 114 | 135 | 11.5 | 6500 | 8 | 3.6 | 6.8 | 87 | 26 | 0.8 | 0.5 | 24 | 26 | 5 | 1 | 3 | 1 | 1 | 1 | 1 |
| 83 | Chandrasekaran | 58 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 45 | 120 | 153 | 11.5 | 560 | 5 | 3.6 | 7.1 | 86 | 25 | 0.5 | 0.6 | 45 | 40 | 3.3 | 1 | 3 | 2 | 1 | 3 | 1 |
| 84 | Hiaun Aung | 42 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 3 | 45 | 125 | 134 | 11 | 4520 | 8 | 3.4 | 6.5 | 88 | 25 | 0.6 | 0.5 | 24 | 28 | 3.5 | 1 | 2 | 1 | 1 | 1 | 1 |
| 85 | Malini Raman | 74 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 41 | 154 | 123 | 9.8 | 6500 | 12 | 3.2 | 7.2 | 86 | 25 | 0.5 | 0.6 | 35 | 30 | 3.4 | 2 | 2 | 1 | 2 | 2 | 1 |
| 86 | Narasimha Samy | 54 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 3 | 2 | 41 | 99 | 121 | 11.4 | 6500 | 20 | 3.6 | 7.1 | 87 | 25 | 0.6 | 0.9 | 25 | 29 | 4.6 | 1 | 1 | 2 | 1 | 1 | 1 |
| 87 | Srinivasalu | 60 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 3 | 3 | 48 | 101 | 121 | 11.5 | 9800 | 5 | 3.1 | 7.2 | 86 | 24 | 0.7 | 0.4 | 24 | 54 | 4.7 | 1 | 2 | 1 | 1 | 1 | 1 |
| 88 | Mariam | 48 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 3 | 45 | 98 | 124 | 10.1 | 6500 | 8 | 3.4 | 7.2 | 110 | 24 | 0.6 | 0.9 | 87 | 89 | 4 | 2 | 2 | 1 | 1 | 1 | 1 |
| 89 | Rajagopal | 81 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 1 | 1 | 2 | 1 | 1 | 2 | 2 | 2 | 51 | 120 | 98 | 10.6 | 7540 | 6 | 3.1 | 7.2 | 99 | 25 | 0.7 | 0.6 | 65 | 69 | 4.6 | 1 | 1 | 2 | 1 | 2 | 1 |
| 90 | Prabakaran | 54 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 35 | 112 | 145 | 9.8 | 6500 | 8 | 3.5 | 8 | 157 | 25 | 0.4 | 0.5 | 25 | 28 | 4.7 | 1 | 1 | 2 | 1 | 1 | 1 |
| 91 | Pichaimuthu | 75 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 3 | 45 | 97 | 114 | 10.8 | 4620 | 10 | 3.6 | 6.8 | 100 | 25 | 0.8 | 0.7 | 80 | 90 | 3.1 | 3 | 3 | 3 | 2 | 2 | 1 |
| 92 | Sugunamei | 50 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 3 | 3 | 45 | 142 | 121 | 10.5 | 4520 | 10 | 3.8 | 8.2 | 124 | 29 | 0.5 | 0.6 | 40 | 42 | 3.1 | 1 | 1 | 3 | 1 | 2 | 1 |
| 93 | Ganesan | 54 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 3 | 54 | 102 | 121 | 11.8 | 9864 | 5 | 3.9 | 6.5 | 98 | 24 | 0.8 | 0.6 | 24 | 28 | 4 | 1 | 2 | 1 | 1 | 1 | 1 |
| 94 | Shafi Ahmed | 55 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 45 | 100 | 120 | 11.9 | 6500 | 5 | 3.5 | 6.5 | 98 | 25 | 0.6 | 0.5 | 35 | 36 | 4.3 | 2 | 2 | 1 | 1 | 1 | 1 |
| 95 | Mohan | 45 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 2 | 3 | 3 | 45 | 132 | 100 | 11.5 | 5632 | 5 | 3.2 | 6.8 | 110 | 25 | 0.6 | 0.5 | 80 | 45 | 4.7 | 2 | 1 | 2 | 1 | 1 | 1 |
| 96 | Ignatius Mary | 68 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 2 | 41 | 100 | 100 | 10.4 | 7430 | 8 | 3.9 | 6.9 | 102 | 25 | 0.6 | 0.9 | 40 | 36 | 3.3 | 2 | 2 | 1 | 1 | 3 | 1 |
| 97 | Balambal | 40 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 3 | 3 | 54 | 98 | 100 | 11 | 8540 | 8 | 4 | 6.8 | 98 | 24 | 0.6 | 0.7 | 80 | 45 | 4.2 | 1 | 2 | 2 | 1 | 1 | 1 |
| 98 | Rajeshwari | 54 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 48 | 100 | 132 | 11.5 | 6800 | 12 | 3.9 | 6.8 | 87 | 24 | 0.6 | 0.5 | 40 | 42 | 3.3 | 2 | 1 | 3 | 2 | 3 | 1 |
| 99 | Radha | 40 | 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 1 | 2 | 2 | 35 | 102 | 120 | 9.8 | 7800 | 12 | 3.4 | 7 | 110 | 28 | 0.5 | 0.6 | 24 | 28 | 4.3 | 1 | 1 | 2 | 2 | 1 | 1 |
| 100 | Kalyani | 56 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 3 | 2 | 41 | 102 | 102 | 10.5 | 4562 | 5 | 3.5 | 6.5 | 98 | 25 | 0.6 | 0.7 | 40 | 45 | 4.3 | 1 | 1 | 2 | 1 | 1 | 1 |
| 101 | Amritha | 48 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 3 | 2 | 41 | 102 | 142 | 11 | 6500 | 5 | 2.8 | 7.1 | 85 | 24 | 0.6 | 0.6 | 25 | 35 | 3.8 | 2 | 1 | 1 | 1 | 1 | 1 |
| 102 | Soundarya | 65 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 48 | 98 | 124 | 10.8 | 7500 | 12 | 3.5 | 8.5 | 145 | 29 | 0.8 | 0.6 | 28 | 32 | 3.5 | 2 | 2 | 1 | 2 | 1 | 1 |
| 103 | Dinesh | 42 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 3 | 3 | 54 | 120 | 145 | 11.4 | 7500 | 8 | 3.6 | 7 | 104 | 32 | 0.8 | 0.6 | 32 | 36 | 4 | 1 | 3 | 1 | 1 | 1 | 1 |
| 104 | Karthick | 54 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 3 | 48 | 124 | 135 | 11.2 | 6500 | 7 | 3.8 | 7.1 | 109 | 30 | 0.8 | 0.5 | 40 | 40 | 3.3 | 1 | 3 | 2 | 1 | 3 | 1 |
| 105 | Subash | 67 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 3 | 41 | 107 | 129 | 11.6 | 4500 | 9 | 3.5 | 7.2 | 104 | 28 | 0.4 | 0.5 | 44 | 48 | 3.9 | 2 | 1 | 2 | 1 | 1 | 1 |
| 106 | Kuppammal | 54 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 2 | 51 | 130 | 167 | 10.6 | 7400 | 8 | 3.6 | 6.5 | 100 | 24 | 0.8 | 0.6 | 28 | 32 | 4 | 1 | 2 | 1 | 1 | 2 | 1 |
| 107 | Bhuvana | 60 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 50 | 145 | 165 | 9.8 | 6500 | 12 | 3.8 | 7.4 | 102 | 20 | 0.5 | 0.6 | 28 | 32 | 3.4 | 2 | 2 | 1 | 1 | 3 | 1 |
| 108 | Shankar | 62 | 1 | 0 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 3 | 2 | 45 | 124 | 168 | 11.8 | 7500 | 12 | 3.6 | 7.2 | 99 | 24 | 0.5 | 0.8 | 36 | 40 | 3.9 | 2 | 1 | 2 | 1 | 1 | 1 |
| 109 | Hemnath | 58 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 3 | 3 | 48 | 135 | 146 | 11.4 | 4500 | 8 | 3.5 | 7.4 | 98 | 25 | 0.5 | 0.6 | 80 | 85 | 4 | 2 | 1 | 1 | 1 | 1 | 1 |
| 110 | Sundarraj | 40 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 44 | 125 | 145 | 10.8 | 6500 | 20 | 3.8 | 8 | 140 | 25 | 0.6 | 0.4 | 40 | 36 | 3.1 | 1 | 3 | 2 | 1 | 3 | 2 |

# KEY

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|---|---|---|---|
| DM | Diabetes Mellitus | SOB | Shortness of Breath |
| HT | Hypertension | HDL | High density lipoprotein |
| IHD | Ischemic Heart Disease | LDL | Low density lipoprotein |
| PVD | Peripheral Vascular Disease | TGL | Triglycerides |
| BMI | Body Mass Index | Hb | Hemoglobin |
| FBS | Fasting Blood Sugar | TC | Total Count |
| BUN | Blood Urea Nitrogen | ESR | Erythrocyte Sedimentation Rate |
| SGOT | Serum glutamic oxaloacetic transaminase | SGPT | Serum glutamic pyruvate transaminase |
| RCA | Right Coronary Artery | Celiac | Celiac Axis |
| LAD | Left Anterior Descending Artery | SMA | Superior Mesenteric Artery |
| LCX | Left Circumflex Artery | IMA | Inferior Mesenteric Artery |